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ORTHOPTIC EXERCISES IN ACHIEVING POSTOPERATIVE THIRD DEGREE FUSION

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THE value of fusion exercises in the treatment of squint has long been a controversial subject, largely because some of the earlier reports led us to believe that strabismus could be fully and permanently corrected and stereopsis established in a large percentage of cases by such treatment alone.¹⁰ However, most students of the subject today recognize that orthoptics generally have little value in correcting the deviation but may be useful in diagnosis and in developing fusion in selected cases.

The purpose of this paper is to appraise the effect of such treatment in achieving a postoperative functional result when the influence of the visual acuity, the age at onset, the duration of the squint and the amount of residual deviation are considered.

During the past few years several excellent reports have appeared on the results obtained with and without the use of orthoptic exercises, in which the percentage of postoperative functional cures varied from 25 per cent to 71.2 per cent (table I).^{2,5,9,13,15,19-21}

MATERIAL

The material for this study was collected from a survey of 256 patients who

underwent surgical treatment for various types of squint. The survey illustrated that a visual acuity of 20/50 or worse in the amblyopic eye prevented postoperative stereopsis on the tests used.³ Therefore, the 80 patients with amblyopia and a visual acuity of 20/50 or worse were excluded, leaving 176 patients with 20/40 vision, or better, for study, 138 of whom had constant squint and 38 intermittent squint.

For the group with constant squint the percentage of functional results seemed to depend somewhat on the presence of hypertropia. Third degree fusion developed in 31.5 per cent of the cases of esotropia uncomplicated by hypertropia, in 25 per cent of those cases associated with overaction of one inferior oblique, in 19 per cent of those cases in which both inferior obliques were overactive, but in only 6.2 per cent of the cases in which the deviation was mostly vertical (table II). Thus it would seem that preoperative hypertropia had a deleterious influence on the percentage of functional results achieved in constant squint.

However, when the deviation was intermittent the presence of hypertropia had little or no effect on the percentage of functional results obtained after surgical operation. Inasmuch as 73.6 per cent of the group with intermittent squint and only 23.2 per cent of the group with constant squint developed a functional cure, it is obvious that the

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TABLE I

REPORTS ON FUSION AFTER OPERATION FOR SQUINT WITH AND WITHOUT ORTHOPTICS

AUTHOR	YEAR	NO. OF CASES	TYPE OF SQUINT	PERCENTAGE WITH FUSION	ORTHOPTICS
Lyle & Foley ¹⁸	1957	287	Esotropia	34.0	Some
Nordlöw ²²	1956	70	Esotropia	59.4	No
Cooper ⁸	1955	382	Esotropia	27.2	Some
		101	Exotropia	71.2	Some
Law ¹⁴	1954	35	Esotropia	45.7	Yes
Alvaro, et al ¹	1954	567	Esotropia and exotropia	71.2	Yes
Scobee ²³	1952	171	Esotropia	42.1	Yes?
Cashell ^{*6}	1952	1119	Esotropia	33.4	Yes
Lyle ¹⁷	1952	144	Esotropia	33.3	Some
Houlton ^{*12}	1952	104	Alternating esotropia	25.0	Yes
Bridgeman ⁴	1952	497	Esotropia	33.8	Yes
Dunnington & Regan ¹¹	1950	79	Esotropia	25.0	No

*Intermittent squint excluded

TABLE II

TYPE OF DEVIATION IN 176 PATIENTS WITH CONSTANT AND INTERMITTENT SQUINT WITH GOOD VISION IN EACH EYE

TYPE OF SQUINT	CONSTANT SQUINT		INTERMITTENT SQUINT		TOTAL NO. OF CASES
	NO. OF CASES	% WITH 3° FUSION	NO. OF CASES	% WITH 3° FUSION	
1. ST without HT	54	31.5	5	60.0	59
2. ST with +IO ₁	24	25.0	3	100.0	27
3. ST with +IO ₂	42	19.0	5	80.0	47
4. XT without HT	2	0.0	11	81.8	13
5. Assorted HT	16	6.2	14	64.2	30
Totals	138	23.2	38	73.6	176
No. with 3° Fusion	32		28		60
No. with 3° Fusion	23.2		73.6		34.1

ST — esotropia

HT — hypertropia

+IO₁ — overaction of one inferior oblique muscle+IO₂ — overaction of both inferior oblique muscles

XT — exotropia

prognosis for a functional result is much better when the squint is intermittent in nature.

METHOD OF STUDY

The eyes of all the children were refracted under atropine cycloplegia at least once before operation, and full or almost full correction was ordered when indicated. In patients with monocular squint, the fixating eye was occluded for months until further improvement seemed hopeless. In patients with alternating squint, alternate occlusion was ordered to prevent suppression and anomalous correspondence or to increase extraocular motility in the patients with limited abduction. Most patients were seen five or six times before an operation was performed.

The amount of surgical correction to be done was gauged by the amount of squint present and was usually divided between the two eyes for the larger deviations, especially when the squint was alternating. Hypertropia of 8 prism diopters or more in adduction was usually corrected along with the horizontal deviation at the time of operation. All patients who had had an operation for squint were excluded.

Special attention was paid to the age of the child at onset of strabismus, the visual acuity and the nature of the deviation (constant or intermittent). The deviation was measured before and after operation by means of prisms and the cover test at six meters and also at 33 cm. with and without glasses in the primary position and also in eyes right, eyes left, eyes up, and eyes down at 33 cm. Bifocals were ordered when indicated. When the postoperative deviation (a phoria or tropia) measured 10 prism diopters or less and the hypertropia measured 2 prism diopters or less in the primary position, the squint was considered fully corrected; but when the postoperative measurements exceeded these limits, the case was classified as a residual postoperative deviation.

TESTS FOR FUSION

At least three tests for postoperative fusion were made at each postoperative visit, using first the Worth four dot test for distance and near. If the patient failed this test, then he also failed the tests for stereopsis. To test for paramacular third degree fusion, the Javal arrow test stereogram (fig. 1) was placed in the stereoscope and the patient was asked to indicate the direction in which

E 7



(Javal 6)

FIG. 1—Arrow test for paramacular fusion. When this card is placed in a stereoscope, a patient with paramacular stereopsis sees a 2-arrow weather vane, in which the top arrow points in one direction and the bottom arrow points at an angle of 90 degrees to the first. When suppression exists, one arrow always appears longer.

the arrows pointed. If stereopsis was present, he saw two arrows of equal length in the shape of a weather vane and could correctly indicate with his finger the direction in which the arrows pointed, i.e., at an angle of 90 degrees to each other. If suppression was present, both arrows appeared to be pointing in the same direction, and one arrow appeared to be longer than the other. As

coming progressively less from line one (20/200 fusion) to line 10 (20/15 fusion).

Of the 60 patients who had stereoscopic vision when last seen, 10 were able to fuse the arrow test only, 44 fused the DB card, and 6 had third degree fusion on other tests (table III). Of the 44 patients with fusion on the DB card, all of whom had a visual acuity of

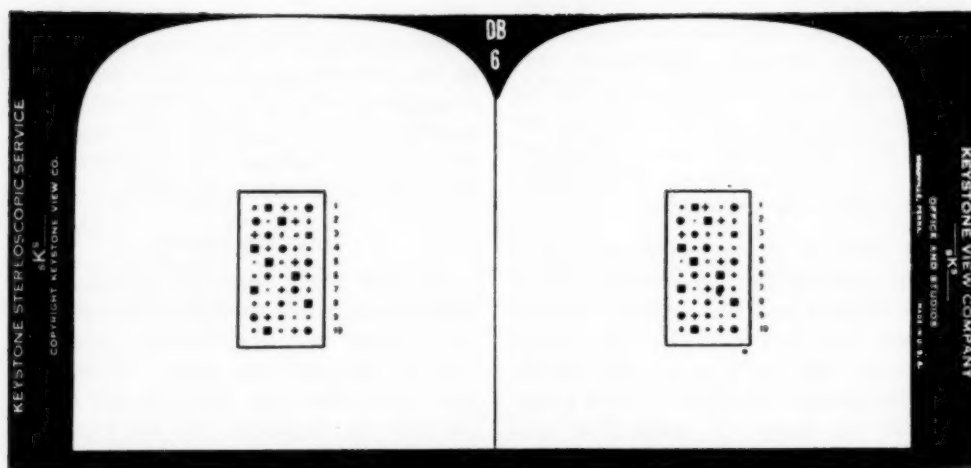


FIG. 2—Keystone test for macular fusion. When this card is placed in a stereoscope, a patient with macular stereopsis sees one of the five characters on each line much closer to him than the rest. The degree of stereoscopic vision is graded from 20/200 in line 1 to 20/15 in line 10.

a check on the patient's responses, the card could be replaced upside down, in which case the direction of the arrows would be reversed. The fly test with polaroid glasses or some other stereograms were occasionally used for this purpose.

The Keystone DB card no. 6-33781 (fig. 2) was next placed in the stereoscope to test for macular stereopsis. When third degree macular fusion was present, one of the five characters on each line stood out from the rest and appeared closer to the patient than all the rest, the forward displacement be-

20/30 or better in each eye, 28 had 20/30 to 20/15 macular stereopsis.

In no case was stereopsis assumed to be present from the appearance of the eyes alone. When necessary, the younger children were recalled and the tests repeated every six to twelve months until they were old enough to understand what was wanted of them. By this means 93.3 per cent of the 60 patients with third degree fusion were kept under observation one year or more, the shortest follow-up being six months (two patients), the longest 19.5 years (one patient) and the average 5.25

TABLE III

TYPE OF POSTOPERATIVE THIRD DEGREE FUSION FOUND IN 60 PATIENTS WITH
CONSTANT AND INTERMITTENT SQUINT

TYPE OF SQUINT	NO. OF CASES	TYPE OF THIRD DEGREE FUSION				
		ARROWS ONLY*	20/200 DB†	20/70-20/40 DB†	20/30-20/15 DB†	FLY, PAIL, OTHER
1. ST without HT	20	5	1	3	8	3
2. ST with +IO ₁	9	0	1	4	3	1
3. ST with +IO ₂	12	3	1	2	5	1
4. XT without HT	9	0	2	0	6	1
5. Assorted HT	10	2	1	1	6	0
Total	60	10	6	10	28	6
Percentages		16.7	10.0	16.7	46.6	10.0

*Paramacular fusion as recorded on Javal stereopsis card (fig. 1).

†Macular fusion as found on Keystone DB-6 stereopsis card (fig. 2).

years. When last seen, the age of the youngest was 5.5 years (two patients), of the oldest, 52 years (one patient); and the average age was 13.75 years. For the remaining 116 patients without post-operative stereopsis, the average follow-up was 4.7 years and the average age 13.8 years.

About 25 per cent of school children between 5 and 7 years old failed the DB card test in a school survey (table IV)

when visual acuity and the binocular reflexes were ignored. A similar survey of children with normal vision and with straight eyes seen in private practice showed that 20 per cent failed the arrow test when age 5, but only 5 per cent did so when age 7; and that 22 per cent failed the Keystone stereogram at age 7, but only 5 per cent did so later in life. Thus, since age was an obvious factor in the percentage of failures, perhaps some patients who failed these tests because

TABLE IV

STEREOPSIS IN SCHOOL CHILDREN AS DETERMINED ON THE KEYSTONE DB CARD*

GRADE	NO. OF PUPILS	NO. FAILED	% OF FAILURE	APPROX. AGE
Kindergarten	423	98	23.2	5
1st Grade	467	129	27.6	6
2nd Grade	411	109	26.5	7
3rd Grade	467	85	18.2	8
4th Grade	452	95	20.9	9
5th Grade	428	78	18.2	10
Total	2648	594	22.4	5-11

*The data in this table, furnished by J. P. H. Jensen, Director of Research, Keystone View Co., were based on a survey conducted in Kansas schools.

of age may develop third degree fusion on these tests later.

The stereoscope, rather than the major amblyoscope, was used to test for stereopsis because it is quicker and easier to use and because I believe it gives fewer false positive results.

NUMBER OF ORTHOPTIC EXERCISES GIVEN

Of the 176 patients, 86 were given orthoptic exercises. The selection of patients to receive exercises was unbiased and mostly a matter of chance because the final decision was generally left to the parents.

The exercises were usually given on the major amblyoscope and were supplemented by home exercises for a number of weeks or months, depending on the progress made. Of the 86 patients given exercises, 11 received the exercises before, 36 after, and 39 both before and after operation. Because the functional results varied so little (from 41 per cent to 45.4 per cent) for these three groups, it would seem that the percentage of functional results achieved by fusion training did not depend on when the exercises were given, i.e., before or after operation.

In order to make a better appraisal of the value of orthoptic exercises, the 138 constant and the 38 intermittent deviations were considered separately.

Table V shows that 67 of the group with constant squint and 19 of the group with intermittent squint received from three to over sixty orthoptic treatments. Since the percentage of functional results did not increase with the number of exercises given to the group with intermittent squint, it is doubtful if such treatment was helpful in establishing stereopsis in these cases.

Because only 23.1 per cent of the patients with constant squint (table V) developed third degree fusion with less than eleven exercises, and 41.1 per cent did so after thirty or more exercises, it would seem that the percentage of functional results obtained depended on the number of orthoptic exercises given. But this is not a valid conclusion because such exercises were generally discontinued early in the treatment when the prognosis for a functional result seemed poor. Thus, the low percentage of functional cures with few exercises was due more to the nature of the squint itself than to the lack of treatment.

TABLE V
NUMBER OF ORTHOPTIC EXERCISES GIVEN TO 67 PATIENTS WITH
CONSTANT SQUINT AND 19 PATIENTS WITH INTERMITTENT SQUINT

NO. OF EXERCISES GIVEN	CONSTANT SQUINT		INTERMITTENT SQUINT	
	NO. OF CASES	% WITH 3° FUSION	NO. OF CASES	% WITH 3° FUSION
3-10	13	23.1	6	83.3
11-30	37	32.4	5	80.0
31-60+	17	41.1	8	75.0
Total	67	32.8	19	78.9

EFFECT OF ORTHOPTIC EXERCISES
IN CONSTANT SQUINT

Of the 138 patients with constant squint, 48 had a residual horizontal deviation (phoria or tropia) of 10 prism diopters or less and a hypertropia of 2 prism diopters or less, while 90 had a residual squint greater than this in the primary position for 20 feet or at 33 cm.

Table VI shows that none of the 90 patients with a residual deviation developed third degree fusion, even though orthoptic exercises were given to 37 of them. Thus it is obvious that fusion exercises were useless in constant squint when a residual deviation persisted.

However, it would seem that orthoptic exercises had real value in achieving a functional result in constant squint when the deviation was fully corrected, because 73.3 per cent of such patients developed stereopsis when exercises were given, but only 55.5 per cent did so when such treatment was omitted (table VI). However, these two groups were not strictly comparable because the

duration of the squint was not the same in each.

To learn what effect the duration of the squint (DOS) had on the functional results, the period of squinting was calculated by the percentage method according to the following formula suggested by Travers²⁴:

$$\frac{\text{DOS in years}}{\text{Age at operation}} \times 100 = \text{DOS in percentage of patient's lifetime}$$

Thus, if a squint began at birth, the duration would always be 100 per cent; if it began at age 1 and was corrected at age 4, the duration would be three years or 75 per cent; if it began at age 3 and was corrected at age 4, the duration would be one year or 25 per cent.

When the duration was calculated in this way for the 48 patients with fully corrected constant squint listed in table VI, and the effect of orthoptic exercises determined when the duration was 50 per cent or less, it was discovered that 90 per cent developed stereopsis without exercises and that only an additional 4.1 per cent did so when such treatment

TABLE VI
EFFECT OF ORTHOPTICS ON THIRD DEGREE FUSION
IN CONSTANT SQUINT WITH AND WITHOUT A RESIDUAL DEVIATION

CASES	WITH NO RESIDUAL SQUINT	WITH RESIDUAL SQUINT	TOTAL NO. OF CASES
No. Given Orthoptics	30	37	67
a. No. With 3° Fusion	22	0	22
b. % With 3° Fusion	73.3	0.0	32.8
No. Not Given Orthoptics	18	53	71
a. No. With 3° Fusion	10	0	10
b. % With 3° Fusion	55.5	0.0	14.1
Total No. of Cases	48	90	138

was given (table VII). Since the exercises increased the percentage of functional results by so small an amount, it would seem that such treatment had little value for patients with fully corrected constant squint when the duration was 50 per cent or less.

But when the duration was over 50 per cent, only 12.5 per cent developed stereopsis without fusion exercises. This percentage is compared to 54.5 per cent when exercises were given (table VII).

when the squint is fully corrected and the duration long.

EFFECT OF ORTHOPTIC EXERCISES ON INTERMITTENT SQUINT

Of the 38 patients with intermittent squint, 21 were fully corrected and 17 had a residual deviation (phoria or tropia) after operation. Table VIII shows that all of the patients whose squints were fully corrected developed postoperative stereopsis whether or not

TABLE VII
EFFECT OF ORTHOPTICS AND OF DURATION OF THE SQUINT (DOS)
ON THIRD DEGREE FUSION IN FULLY CORRECTED CONSTANT STRABISMUS

CASES	DOS TO 50%	DOS OVER 50%	DOS UNKNOWN	TOTAL NO. OF CASES
No. Given Orthoptics	17	11	2	30
a. No. With 3° Fusion	16	6	0	22
b. % With 3° Fusion	94.1	54.5	0.0	73.3
No. Not Given Orthoptics	10	8	0	18
a. No. With 3° Fusion	9	1	0	10
b. % With 3° Fusion	90	12.5	0.0	55.5

Thus orthoptic exercises appear to have real value in constant squint when the deviation has been fully corrected and had existed a long time. However, a functional cure was never achieved when the squint dated from birth, even though the deviation was fully corrected in 5 patients and extensive orthoptic exercises were given to 2 of these.

In summary, then, it would seem that orthoptic exercises cannot bring about a functional cure in constant squint when a residual deviation is present or when the squint is congenital, and they are of doubtful value when the deviation is fully corrected and of short duration; but such treatment may be of real value

exercises were given. Therefore, the exercises must have been superfluous in these instances of fully corrected intermittent squints.

However, when a residual deviation persisted, 55.5 per cent developed stereopsis with the exercises, but only 25 per cent did so when such treatment was withheld (table VIII). Thus, orthoptic training seemed to be valuable for the patients with a residual postoperative deviation.

When the duration of the squint was considered for all 38 patients with intermittent squint, the percentage of functional cures seemed to be completely independent of how long the deviation

had existed. Hence, a long duration had a much less harmful effect on the percent than in constant squint.

percentage of functional results in intermit-

Briefly then, orthoptic exercises seemed to be unnecessary in the fully corrected intermittent cases but may have been of real value when a residual deviation persisted.

DISCUSSION

After years of controversy regarding the merits of orthoptic exercises in the treatment of squint, and after analysis of thousands of cases given such exercises, the true value of fusion training is beginning to emerge. Lyle,¹⁶ Douglas,⁹ and others believe that such treatment has little or no value in correcting the deviation itself but may be indispensable in building up fusional amplitudes in selected cases. Moreover, Arstikaitis and Condie²² and others emphasize that such exercises are useless in establishing stereopsis unless some third degree fusion existed before operation. The question therefore arises: are orthoptic exercises of real value in achieving a functional result in surgical treatment of squint and, if so, how can such cases be recognized clinically?

This survey has demonstrated that a functional result never occurred when amblyopia of 20/50 or worse was present, when a residual constant squint, as defined above, persisted, or when a constant squint began at birth.

However, a functional cure occurred frequently in constant squint when the vision was 20/40 or better in the amblyopic eye, when the deviation was fully corrected, and when the squint began after birth. Under these conditions orthoptic exercises were useful in developing a functional result in only 6 patients, all of whom had squinted during more than 50 per cent of their lifetime (table VII). In intermittent squint such exercises were useful in establishing a functional cure in only 5 patients, all of whom had a residual deviation after operation (table VIII). Therefore, orthoptic exercises were useful in only 11 patients; and, since at least 3 of these probably would have developed stereopsis had the exercises been omitted, it is estimated that such treatment was useful in only 8 cases, 5 per cent of the 176 cases, or in only 8 or 13.3 per cent of the 60 patients who had postoperative stereopsis on the tests used here. It should be added, however, that this

TABLE VIII
EFFECT OF ORTHOPTICS ON THIRD DEGREE FUSION
IN INTERMITTENT SQUINT WITH AND WITHOUT A RESIDUAL DEVIATION

CASES	WITH NO RESIDUAL SQUINT	WITH RESIDUAL SQUINT	TOTAL NO. OF CASES
No. Given Orthoptics	10	9	19
a. No. With 3° Fusion	10	5	15
b. % With 3° Fusion	100	55.5	79.0
No. Not Given Orthoptics	11	8	19
a. No. With 3° Fusion	11	2	13
b. % With 3° Fusion	100	25.0	68.4
Total No. of Cases	21	17	38

conclusion is not entirely valid statistically because of the few cases in some categories.

Even though orthoptic treatment is of little value in establishing a functional result in squint which requires surgical operation, it may be valuable in determining whether the prognosis for such an operation is favorable, especially when the history of the duration of the squint is uncertain. For example, if a patient has a satisfactory cosmetic result after operation, but has a small residual horizontal or vertical squint sufficient in amount to prevent fusion, a second operation is contraindicated if an orthoptic appraisal reveals little or no fusion; but if this analysis demonstrates the presence of good fusion, the prognosis for a functional result is good and, therefore, the residual squint should be corrected surgically. However, since one can generally predict fairly accurately from the history and the clinical findings which cases have a good prognosis for a functional result and which have not, it is unlikely that an orthoptic analysis on one of the major amblyoscopes is indispensable in surgical treatment of squint.

It is interesting to speculate why some of these patients developed third degree fusion and others did not. For example, why did none of the patients with amblyopia and 20/50 vision develop postoperative stereopsis when other patients without squint and with a visual acuity of 20/50 (due to anisometropia, corneal or lens opacities, or a macular lesion) have been known to do so? By way of explanation one can assume that the patients without squint had fusion before the impaired vision occurred, or that they developed it despite the reduced visual acuity, or that the area of suppression in the patients with squint was so large and deep as to preclude fusion on the tests used.

Also, why did none of the patients with constant squints dating from birth develop a functional result when the vision was good in each eye and the deviation was fully corrected? In explaining such failures, Worth²⁵ postulated a congenitally defective or absent fusion faculty, while Chavasse⁷ attributed these failures to the postnatal development of abnormally conditioned sensory and motor reflexes.

Why were fusion exercises unnecessary for patients with fully corrected constant squint of late onset and of short duration, but apparently helpful when the strabismus occurred very early in life and its duration was long? In explanation one can assume that patients with late onset had good fusion at the time of operation and needed only surgical realignment of their visual axes for fusion to be re-established; whereas, patients with a history of early onset and long duration had a weak fusion ability which was incapable of holding the visual axes parallel after operation but which could be sufficiently augmented by fusion training to convert a small tropia to a phoria.

Why were fusion exercises superfluous for patients with fully corrected intermittent squints, but helpful in the presence of a residual deviation? The answer must depend on a fairly well developed fusion faculty which was adequate to maintain fusion in the fully corrected but not in the undercorrected cases without the benefit of fusion training.

From this survey it is obvious that great strides in achieving a functional result in squint requiring surgical treatment will not depend primarily on giving orthoptic exercises, but rather in directing our efforts toward elimination of amblyopia ex anopsia and toward full correction of the squint soon after the

deviation begins. For example, of the 256 cases, 31 per cent (80 cases) of the functional failures were due to amblyopia, 39 per cent (100 cases) were associated with inadequate correction of the squint and 7 per cent (16 cases) were the result of early onset, a long duration, or some other unknown cause, leaving only 23 per cent (60 cases) with a satisfactory functional result. Thus, the secret of success in obtaining a high percentage of functional cures in surgical treatment of squint must depend on preventing amblyopia ex anopsia and in fully correcting the squint as soon as feasible.

CONCLUSIONS

1. Amblyopia of 20/50 or worse in one eye with normal vision in the other prevented a functional result in constant and intermittent squint even though the deviation was fully corrected.

2. Orthoptic exercises were useless in patients with constant squint when a residual postoperative horizontal deviation of over 10 prism diopters or a hypertropia of over 2 prism diopters existed in the primary position.

3. Orthoptic exercises were of little value in establishing a functional result in patients with constant squint when the deviation had been fully corrected and the squint had existed less than 50 per cent of the patient's lifetime, because almost as many developed third degree fusion without as with such treatment; but these exercises may have been valuable for patients with constant squint when the deviation was fully corrected and had been present for more than 50 per cent (but less than 100 per cent) of the patient's lifetime.

4. Orthoptic exercises were unnecessary in the establishment of a functional result in patients with intermittent

squint when the deviation was fully corrected, because 100 per cent of such patients developed third degree fusion without exercises; but the treatment appeared to be useful in treating intermittent squint when a residual deviation persisted.

5. The prognosis for a functional result, with or without orthoptic exercises, was always better in intermittent squint than in constant squint.

6. Patients with constant squint dating from birth did not obtain postoperative third degree fusion, even though the squint was fully corrected, the vision was good in each eye, and extensive orthoptic exercises were given.

7. Orthoptic exercises were indispensable in obtaining a functional result in only 5 per cent of 176 patients treated surgically for various types of squint with good vision in each eye. The remaining 95 per cent either developed third degree fusion spontaneously after operation or failed to do so because of onset of the squint at birth, a long duration of the squint, or a residual deviation.

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OCCLUSION OF THE PARAMACULAR IMAGE IN THE DEVIATING EYE

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VARIOUS types of occlusion have been used for amblyopia, suppression, and abnormal sensorial relationships in association with, or as the result of, strabismus.

At the turn of the century, occlusion was used by Worth,³ Javal, and others, although comparatively little was known about the possibilities of the cortical re-education of binocular functions.

Full occlusion of the amblyopic or eccentrically fixing eye was used accidentally on a few occasions. It is effective in certain instances. A number of years ago, in checking the visual acuity of a patient, a group of technicians and I found that the acuity of the covered eye was 20/70, while that of the uncovered eye read 20/20. In checking the previous notes on this patient, we found that acuity of the covered eye had previously been 20/200. After ten weeks of total occlusion, there was actually an improvement in the visual acuity of the amblyopic eye. This seems completely contradictory to the theory of redeveloping the usefulness of an amblyopic eye by constant use, as is done by constant occlusion of the dominant eye. In this case, it was not the constant use of the eye, but rather the

exclusion of distracting images and the complete rest which should be credited for the improvement.

Not only is suppression amblyopia found, but nonamblyopic suppression, or suppression with relatively good visual acuity, is also a rather common finding in association with strabismus. Webster defines suppression as a "forcible exclusion of something from conscious activity." Adler¹ describes it as a psychological function, "an inhibitory reflex." He also says:

Conditioned reflexes are formed, in general, when a stimulus, which by itself causes no response, is repeatedly associated with another stimulus which *does* evoke a response.

With regard to vision, he says:

Suppression is conditioned by the use of the two eyes simultaneously; whereas amblyopia is a nonconditioned loss of acuity, which remains when one or both eyes are being used.

If, then, suppression and amblyopia are produced differently, perhaps they should be treated differently. We have found that some patients with amblyopia are well able to maintain both the right and left images in consciousness when working with them on the troposcope. There are those, however, who completely suppress one image while the other eye is fixating. Patients with fairly equal vision, and also with phorias, frequently speak of one image disappearing when both maculas are properly focused on the object.

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The relatively new type of half-lens occlusion is exceedingly helpful in reducing some types of amblyopia and some types of suppression, and in eliminating abnormal sensorial relationships. With these three factors of suppression in mind, I shall attempt to justify half-lens occlusion.

Nonamblyopic suppression is a phenomenon occurring only when both eyes are used simultaneously. Therefore, it must also be eliminated simultaneously, or the reflex must be reconditioned with both eyes simultaneously in use. We must eliminate the cause for the forcible exclusion from conscious activity by introducing a new situation in which we can guide the responses into the proper channels. We know that an image or light stimulus falling on the retina, either temporally or nasally to the macula, in one eye is a stimulus which evokes the necessity of eliminating or suppressing one image. Unless the deviation is small, the image of the deviating eye falls on the retina parafoveal to the point of acute sensitivity except for form. Therefore, it is as easily acceptable that the other stimulus may be an eccentric beam of light, or the second image of the object fixated.

Regarding abnormal sensorial relationships, Dr. Burian² says:

The fovea of one eye and an eccentric retinal element of the other eye . . . acquire a common visual direction. This new sensorial relationship is termed anomalous, and corresponding retinal elements are those elements of the two retinæ, the stimulation of which, in binocular vision, give rise to the localization in one and the same visual direction, no matter whether the stimulus reaches the retinal elements in one eye alone or the corresponding elements in the other eye alone, or both simultaneously.

The half-lens occlusion is designed *and measured* to eliminate the eccentric beam of light on the retina, which is the source of the aberrant stimulus, the stimulus which evokes suppression. The

displaced corneal reflection is the guide by which this occlusion is measured. The occlusion must be applied on the temporal side of the lens if the corneal reflection is temporally displaced, as in esotropia. It must be applied to the nasal side of the lens if the reflection is nasally displaced, as in exotropia. The size of the occluding material must be carefully measured by the technician to block off this reflection or, preferably, one-half of the pupil. Occlusion of half the lens would certainly not be adequate for 40 to 50 diopters of esotropia, nor would a half-occluded lens be proper for 10 diopters of exotropia. When the deviation is greater for distant fixation, the gaze must be directed to a distant fixation target and the occluder applied to cover the deviated corneal reflection. When the deviation is greater for near, it must be measured with near fixation. The former application is more frequently indicated in exotropia, or in divergence excess.

Success lies not only in removing the distracting stimulus by occlusion itself, nor even in its proper application, but in the subject's proper use of the occlusion. If a child continues to use the dominant eye without any recognition of the dormant macula, half-lens occlusion is not really effective. When this occlusion has been applied, the child must be taught (1) that both eyes can see, and (2) that both eyes can see simultaneously. He should not be taught to think in terms of *which* eye sees, but rather in terms of *what* can be seen in space. We must help the child to reorient and re-educate his sensorial responses to localize the two images in proper space relationship, so that eventually the two maculas again have a common visual direction.

To achieve this goal, the child must learn to fixate from one image to the other without blinking, first by alternate

cover, then by covering and uncovering, then by using a septum. Finally, the child must *feel* alternate fixation movements of the eyes. To enhance the appreciation of diplopia, a red filter can be used advantageously to help the child recognize the proper sensorial relationship of the two images in space. When diplopia is first seen and appreciated by the child, there need be no doubt in the technician's mind as to the reliability of the response. A sudden expression of unbelievable surprise comes over the child's countenance, even without a word spoken, when the two images are recognized; and once recognized, they are also interpreted.

To carry on at home the practice of recognizing diplopia, the child can use the filter while watching television. It should be used over the dominant eye because it decreases the brightness of the television screen, thus making sure that the deviating eye is used to fixate the clear screen.

If, and when, the child has effectively been taught the use of this type of occlusion, he should begin to fixate constantly with the nondominant eye. In effect, he should turn his face slightly toward the side of the occluder and persuade himself to use the macula of that eye, while the dominant eye deviates. If this is done correctly, the suppressed area decreases and the macula becomes active. The nondominant macula is used in active relationship to the other macula in the office under active training, also at home when he watches television, and every time the parents take time to alert the child to diplopia for any casual seeing. When the child has learned to appreciate diplopia at the office and for television, he should also be able to recognize diplopia of any object or person, at least when using a septum. The hand, held at right angles to the face, serves well

as a septum. First, the child is taught to recognize diplopia when looking at the technician, then to see two mothers; after that, it can be carried over to other objects in the room.

Anomalous retinal correspondence is *believed* less prevalent in divergent strabismus. The high deviations show a large area of suppression, and occasionally it is difficult to teach the child to recognize heteronymous diplopia, no matter how far the images are separated, or which method of stimulation is instituted. When nasal occlusion is applied and the head is turned so that the nondominant eye fixates, the child recognizes the movement of the eyes in fixation more easily and simultaneously recognizes the correct spatial relationship.

A vertical component is frequently found in association with the lateral deviation. This complication seems to make recognition of diplopia more difficult than it is for a lateral deviation alone. A three-quarter patch can be used to cover the lower half of the lens if the deviating eye is also the hypertropic eye. The change from primary position to reading position creates a handicap. The deviating, or suppressing, eye in such cases should always be occluded for reading. We now use base up and base down prisms in clip-over frames to neutralize the vertical deviation as nearly as possible. With this aid, the child is more easily aware of the lateral diplopia and will utilize the nasal or temporal occlusion properly. We have relatively few complaints about the occluder detracting from the child's appearance, or about discomfort due to the added weight.

I prefer to break down suppression and teach the child to fuse before the original suppression areas are altered by surgical operation. I favor early

surgical operation only when the deviation is so large, 60 prism diopters of esotropia or more, that it is not feasible to use the major amblyoscope for orthoptic training. Most of the patients with exotropia who have been referred to me have thus avoided surgical operation, and the patients with esotropia have proved their flexibility of amplitudes. The attempt to break down suppression and teach fusion can furnish further evidence of the necessity for surgical correction. This method guards against overcorrection, which may be done because of apparent poor convergence amplitude, and helps to determine when full refractive correction is necessary to permit adequate relaxation for distance fixation.

SUMMARY

Half-lens occlusion is an effective means of active stimulation in cases of macular suppression:

1. It blocks out the distracting impulses from the paramacular area of the deviating eye.
2. It makes the patient conscious of the dormant eye, with the dominant eye also working.
3. It stresses diplopia in proper space relationship.
4. It guides diplopia into proper fusion channels.

The results are (1) better visual acuity, (2) elimination of suppression, and (3) proper space relationship; common visual direction; fusion.

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FIXATION DISPARITY AND OCULOMOTOR IMBALANCE

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It is apparent from statements found in the literature^{1,2,4-7,15,17} that the phenomenon of fixation disparity is not generally understood. In an effort to correct this situation, I shall state at the outset what the phenomenon of fixation disparity is not:

First, and this point is emphasized, fixation disparity is not a "small angle squint." Nor does its existence in a patient indicate a latent squint, unless any heterophoria is also accepted as an indication of a latent squint.

Fixation disparity *cannot* be seen by the cover test. Not only is the angle far too small, but, due to the phoria, it would be completely masked by the eye movements.

Fixation disparity is *not* an indication of a functional lack of foveal binocularity.

Fixation disparity is *not* an indication of an anomalous correspondence.

It is *not* possible to differentiate between a heterophoria and a heterotropia on the basis of whether or not fixation disparity exists.

Fixation disparity is *not* related to foveal suppression.

Finally, fixation disparity has no effect on stereoscopic depth perception, nor does it have anything to do with aniseikonia.

The misconceptions that have arisen tend to make the phenomenon more complicated than it actually is. Fixation disparity is a phenomenon of normal binocular vision and exists only when there is oculomotor imbalance. To repeat: fixation disparity will occur only, first, if the subject has some fusion and, second, if there is an oculomotor imbalance or a phoria at the test observation distance.

The phenomenon itself is relatively easy to describe,¹⁰ but some of the most basic concepts of the physiology of binocular vision are required in order to understand it. For the moment, suppose two sets of parallel lines (fig. 1) are observed in a stereoscope (haploscope or amblyoscope) or even by free fixation. The separation of the pair of lines to be seen by the right eye is greater than that of the pair to be seen by the left eye. Except under unusual circumstances, this difference in separation of the two pairs of lines corresponds to a disparity between the images in the two eyes of the lines. In spite of this disparity (if not too great), a normal person will observe only two lines in the stereoscope. Consequently it is said that the images of corresponding lines fuse in the two eyes; each is seen binocularly in one subjective direction.

From the Mayo Clinic and the Mayo Foundation. The Mayo Foundation, Rochester, Minnesota, is a part of the Graduate School of the University of Minnesota.

Presented at the Annual Joint Meeting of the American Orthoptic Council and the American Association of Orthoptic Technicians, Oct. 13, 1957, Chicago.

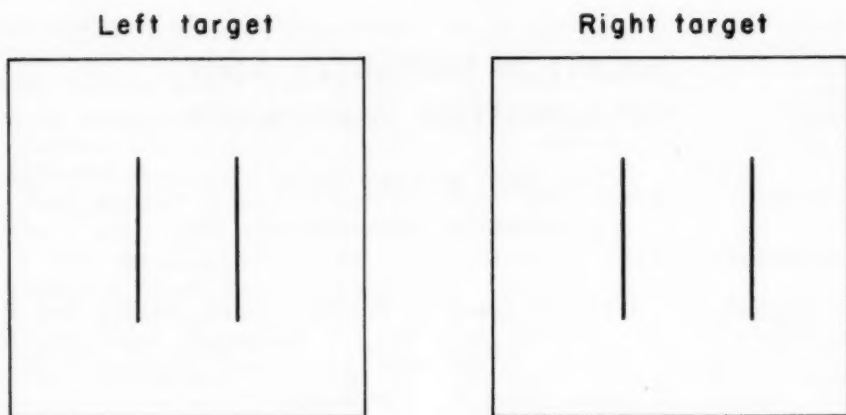


FIG. 1—Paired targets to be used in the stereoscope to illustrate fusion of disparate images.

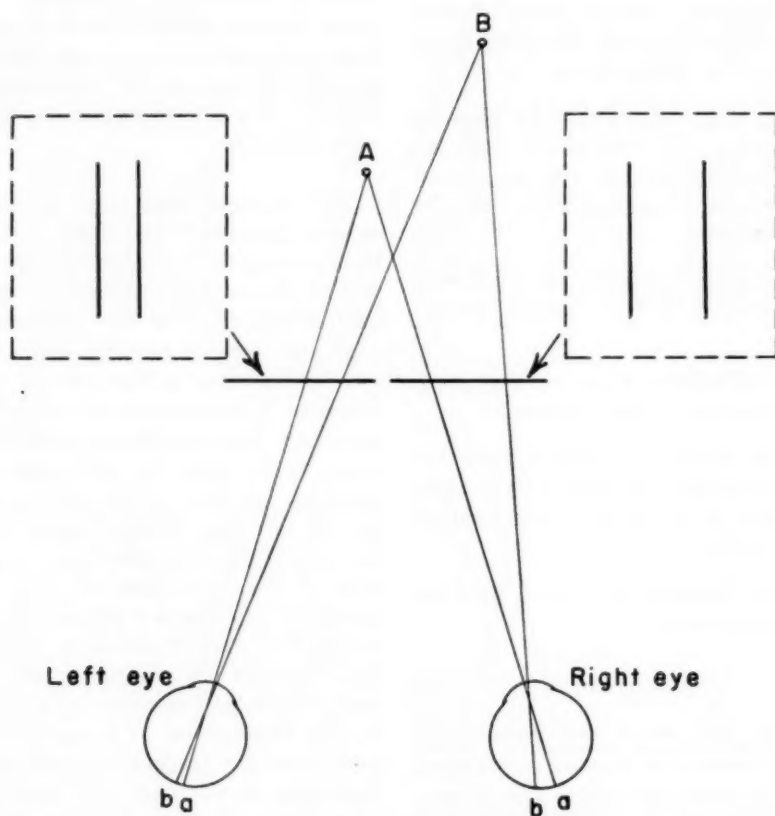


FIG. 2—Geometric relationship between two objects in space and the corresponding targets to be observed binocularly.

The technician also observes (if the right pair of lines is seen by the right eye) that the line on the right appears farther away than does the left by virtue of stereoscopic depth perception (fig. 2). If the left of each pair of lines is fixated, the right of each pair of lines will be imaged in the two eyes in uncrossed disparity.

Experimentation has shown that the separation of one pair of lines can be varied only within certain limits (fig. 3,d) in order that the images of the stereoscopic pair of lines will appear as one, that is to say, seen in one subjective direction. These limits, while somewhat indefinite, determine the horizontal extent of Panum's areas of fusion.

It can be verified that the images of the right line are disparate if suitable control marks are included in the figure (fig. 4). Then the unilocularly seen marks will be seen displaced as one would expect from the difference in separations of the two lines. In the case of marked ocular dominance, the subjective direction of the fused image may appear the same as that of the mark seen by the dominant eye, as shown in A or C of figure 4. If no such marked

dominance exists, both marks will appear displaced, as in B of figure 4, from the subjective visual direction of the fused images. These sketches illustrate that disparate images can be fused with the emergence of a single subjective direction and that the single subjective direction may be a compromise between the two primary unocular directions or may agree with the primary unocular direction of the image in one of the eyes. The images of the guide marks are seen in the primary subjective direction corresponding to the retinal elements on which they fall.

It can be shown also that Panum's areas exist at the foveas—for objects near the fixation point. In figure 5, the eyes fixate the lower tip of a needle (F). A needle (P) can be moved a definite, small distance farther or nearer than F, before double images of P appear. The sum of the small angles of uncrossed and crossed disparity in the two cases defines the horizontal extent of Panum's areas near the fixation point. The angular size of this extent is about 6 to 10 minutes of arc. It is clear, therefore, that, analogous to the fusion of the disparate images in the preceding example, the eyes need not be perfectly

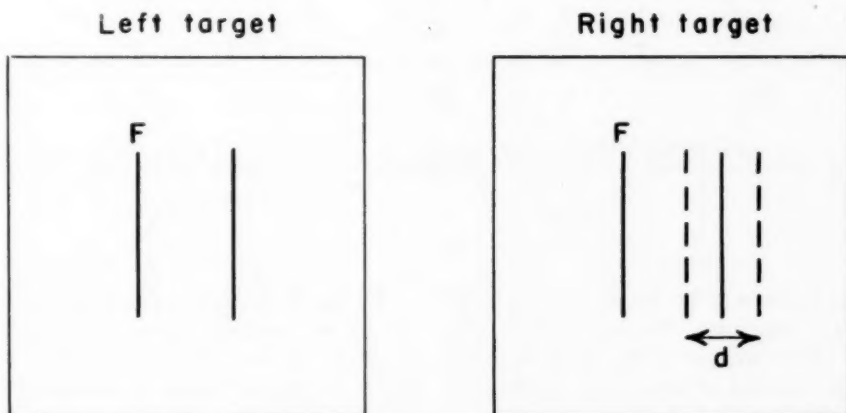


FIG. 3—Targets to illustrate limiting disparities (d) for fusion of images.

converged on the fixation point for the images of that point to be fused.

The nonius horopter experiments¹⁰ show indeed that, if there is an oculomotor imbalance, or a tendency of the eyes to turn out or to turn in (phoria), the eyes will actually underconverge or overconverge by a small angle so that the images of the fixation point will actually be disparate, and yet the images

In order to observe and measure the effect, special targets must be devised for use in a stereoscopic instrument, which have identical details on each so that their images will be able to stimulate fusion, and which also have suitably placed control marks that exert no stimuli for fusion. Many examples of these targets have been described in the literature,¹⁴ and several will be described herein to emphasize certain ideas.

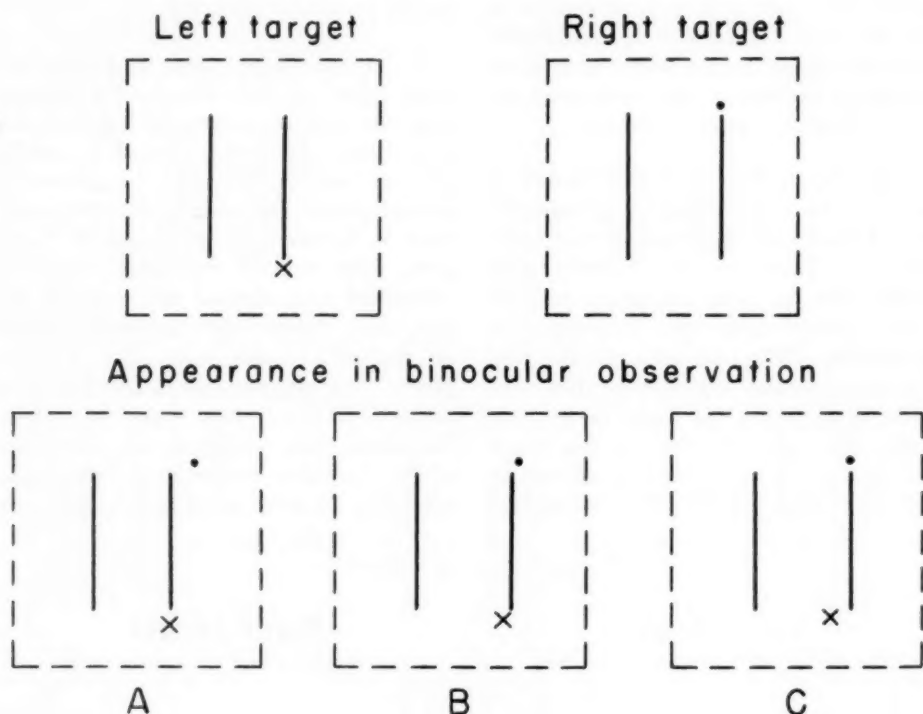


FIG. 4—Same targets as those in figure 1, but with guide or control marks. A, B and C—The three possible ways in which the subjective direction of the fused disparate images may appear relative to the marks (exaggerated).

of that point will be fused, that is, seen in one subjective direction. The disparity of the images of the fixation point will be crossed if the eyes exhibit an exophoria, and uncrossed if an esophoria.

In figure 6, only a single vertical line serves as identical detail for fusion and fixation of the two eyes. The small circle in the left target is drawn close to the line. In the right target the small dot is placed the same distance from

the line as is the center of the circle in the left target. When these two targets are viewed in a haploscope, special attention should be paid to the apparent position of the dot within the circle. Depending on the oculomotor imbalance for the particular optical conditions provided by the haploscope, the dot will appear displaced from the center of the circle—to the right in esophoria, and to the left in exophoria. If no imbalance exists, the dot will be centered in the circle. When strict fixation is directed to the line, the displacement can still be seen. This is evidence that the images of the fixation point are indeed disparate when there is an oculomotor imbalance.

Figure 7 illustrates similarly constructed targets when the fusion details are seen centrally (upper panel) or are seen peripherally (lower panel) and shows the displacement of the unocularly seen portions of the targets caused by an esophoric imbalance. Thus, in order that the phenomenon be observed, certain parts of the targets have to be blocked out in order to insert the unocularly seen test details. The phenomenon operates the same (though perhaps to different extents) regardless of what part of the visual field is blocked out. If there are other object points in the binocular visual field, the disparities of all their images will be increased or decreased by the amount of

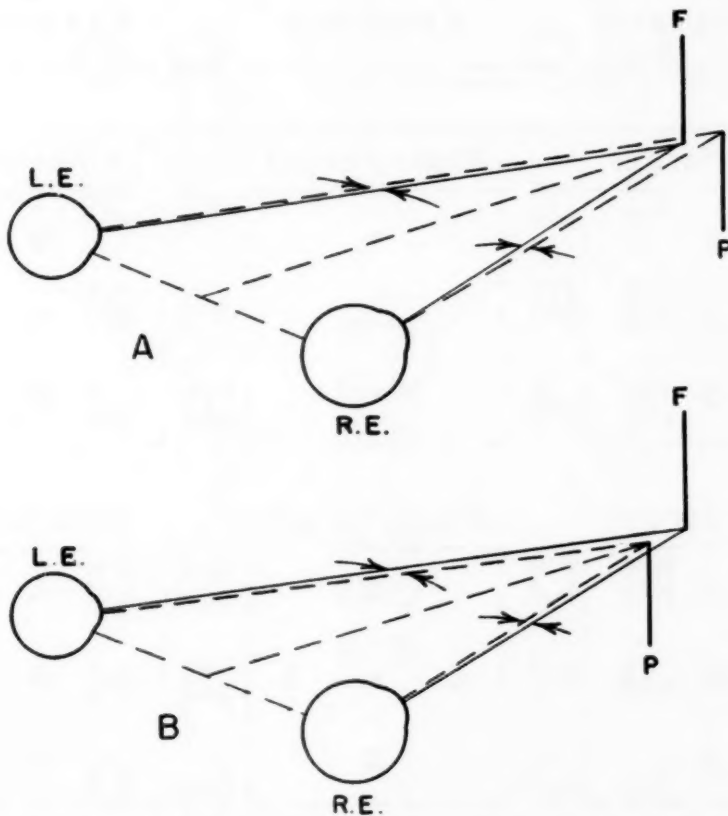


FIG. 5—Perspective sketches to illustrate that Panum's fusional areas exist at the fixation point. See text for explanation.

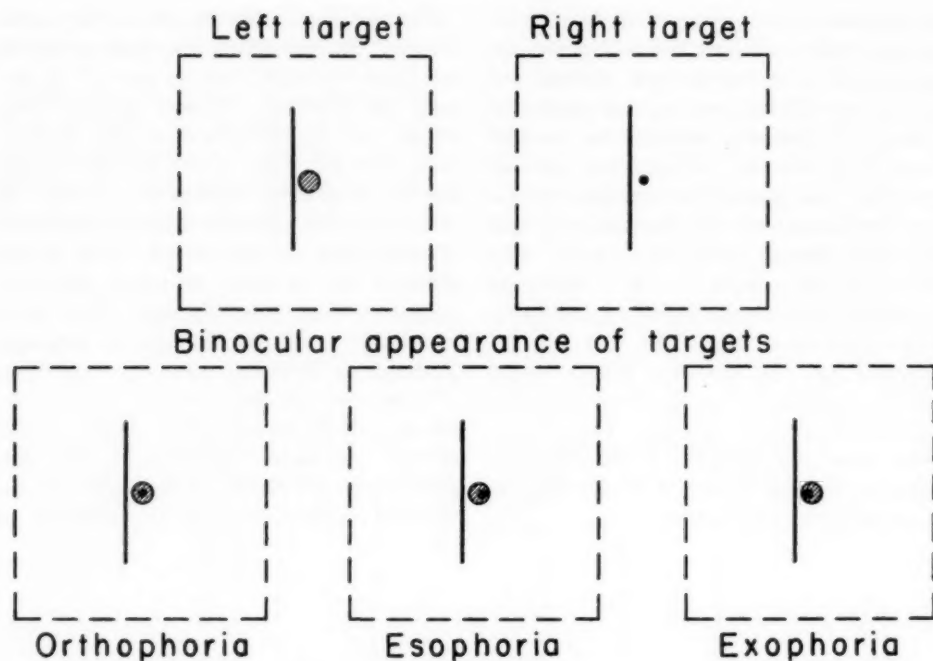


FIG. 6—Targets to be observed in stereoscopic instruments to illustrate fixation disparity with the test details near the line fixated in orthophoria, in esophoria, and in exophoria.

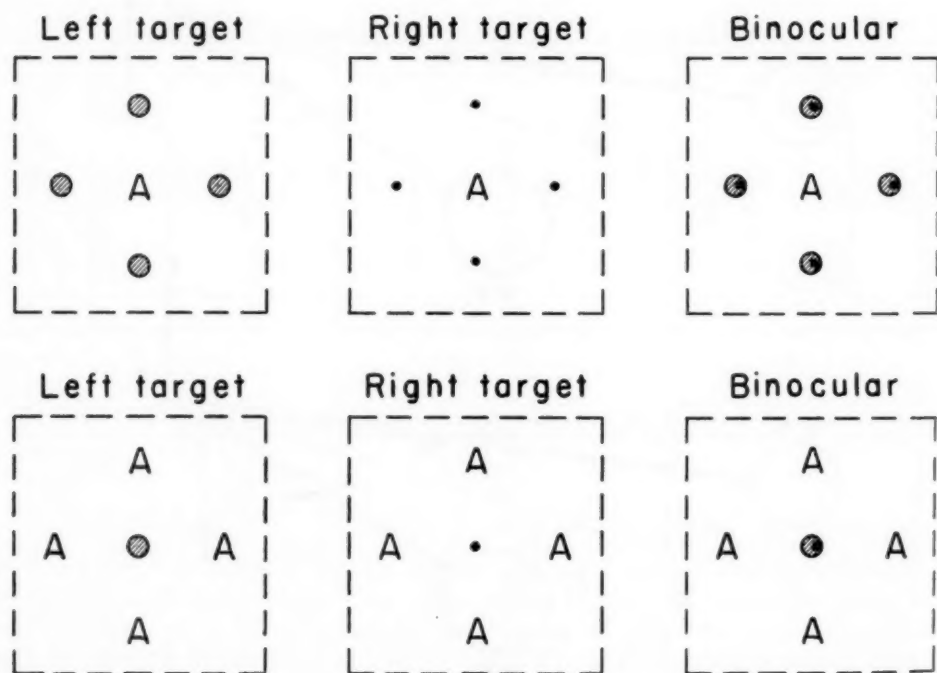


FIG. 7—Targets to be observed in stereoscopic instruments to illustrate a fixation disparity in the periphery and in the central parts of the visual field, in the case of an esophoria.

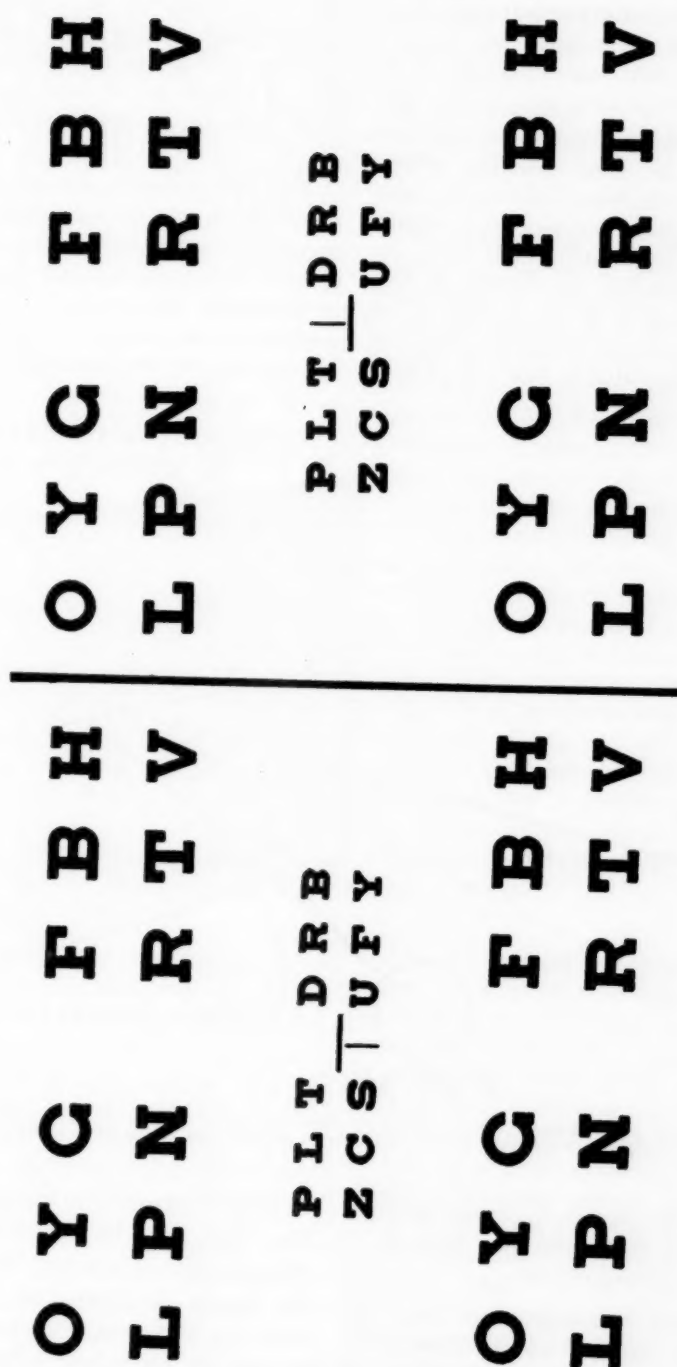


FIG. 8—Targets that can be used in an amblyoscope or a synoptophore to observe fixation disparity.

the disparity of the images of the fixation point. This is the reason that early studies described the phenomenon as a "slip."

To observe or to measure the disparity with maximal precision, the test details must be seen foveally, where visual acuity is highest, but this requires that the details for fusion in the central areas be blocked out. Then fusion is actually maintained by details in the extrafoveal areas in a visual field. One type of target used for observing fixation disparity is illustrated in figure 8.⁹ These

test details are projected by lanterns onto a screen for the distant vision test¹⁴ and are presented as a photographic transparency for the near vision tests.¹² The use of Polaroid film permits separation of details to be seen monocularly from those to be seen binocularly.

The device for presenting the test or nonius lines in the test details has been specially constructed so that the lower of the lines can actually be displaced horizontally relative to the stationary position of the upper line, and the magnitude of any displacement can be ac-

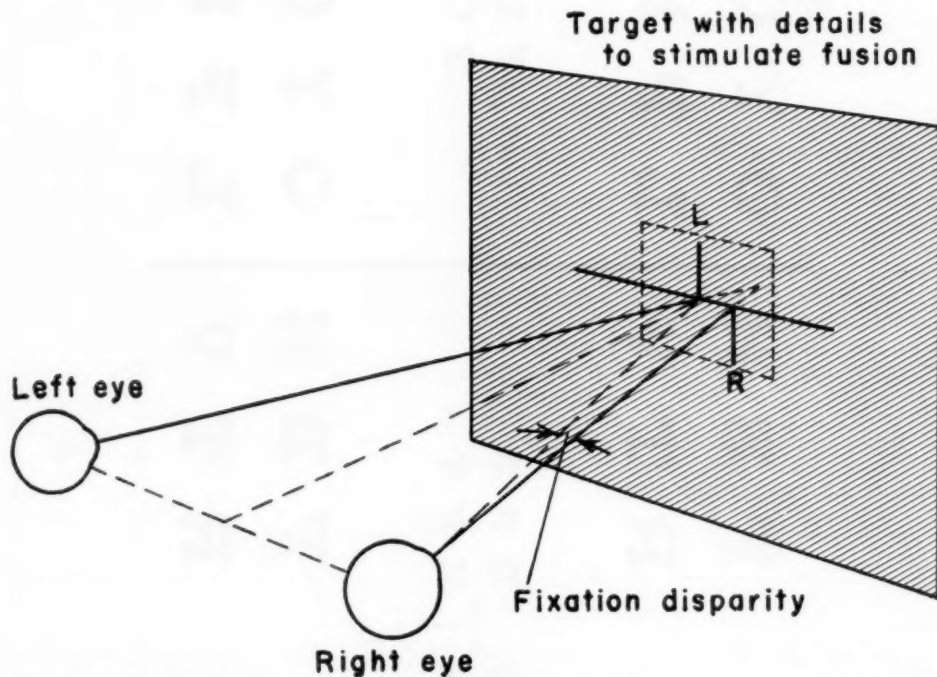


FIG. 9—Perspective sketch showing how the nonius test lines have to be adjusted in a test instrument to appear aligned when an exophoria is present. The distance between the two lines is a measure of the fixation disparity.

figures can be used in the major amblyoscope. The test lines are actually a nonius or vernier device.

To obtain measurements with the eyes under normal visual conditions, rather than under the artificial ones of the common stereoscope, the fusion and

curately measured. In the test the lower line seen only by the left eye is displaced until it appears directly below the upper line seen only by the right eye; that is to say, both lines will then appear in the same subjective visual direction. The actual displacement of

these two nonius lines when they appear aligned is the fixation disparity.

The test details are adjusted to appear one directly below the other for an individual with exophoria (fig. 9). The true convergence of the eyes is behind the target. The two nonius test lines have been adjusted so that they appear aligned—one directly beneath the other. The actual separation of the two lines subtends the small angle that is used as the measure of the fixation disparity. This angle is conveniently expressed in minutes of arc. An observer with normal 20/20 visual acuity can usually discriminate the displacement of the lower line relative to the upper by 1 minute of arc. This is about the precision of measurement except when the oculomotor imbalance becomes large and variable.

The phenomenon of fixation disparity shows that, in spite of the efforts of the fusional convergence to control the pointing of the eyes so that diplopia is prevented, a small error in convergence in the direction of the phoria actually occurs. This indicates that the oculomotor imbalance due to a phoria still exerts a continual effort to turn the eyes toward the phoria position. This small error in convergence is the fixation disparity.

The magnitude of the displacement of the unilocularly seen test details in any given instance depends primarily on the degree of the oculomotor imbalance, but it depends also on the strength of the compulsive reflex for fusion in the targets. When the conditions of observation and measurement are kept constant, the results of all measurements are comparable.

Under such constant conditions the fixation disparity will increase with the increase in the effort required to maintain fusion, and this effort will be related to the difference between the con-

vergence required for fusion and the convergence corresponding to the phoria. An oculomotor imbalance means that a difference exists between the convergence required for fusion and the phoria position (or relative position of rest) for the given observation distance.

An insight into the nature of this fixation disparity is gained when its magnitude is measured as the oculomotor imbalance is altered. The convergence required for fusion can be altered by introducing prisms, base in or base out, before the eyes at a constant observation distance. By placing ophthalmic lenses before the eyes to change the stimulus to accommodation, the convergence corresponding to the phoria can be changed by virtue of the accommodative convergence synkinesis. Both the convergence required for fusion and the stimulus to accommodation will be changed by a change in observation distance. Thus in the normal use of the eyes, with change in observation distance, the convergence required and the accommodative convergence, through the change in the stimulus to accommodation, *tend* to increase together and proportionally.

As an example, suppose measurements of fixation disparity at a near visual distance are made, first, normally and, then, for each of a series of prisms of increasing prism power placed first base in and then base out before the eyes. Suppose the subject is orthophoric at the observation distance, so that the fixation disparity would normally be zero. Now if we put prisms amounting to 2 prism diopters base in before the two eyes (fig. 10), we have decreased the convergence required if fusion is to be obtained by 4 prism diopters, yet the stimulus to accommodation has remained unchanged, so that the phoria

position of the eyes relative to the convergence required is now 4 prism diopters esophoric. An esophoric fixation disparity is then measured. The reverse would be true if the prisms had been placed base out.

A picture of how the fixation disparity changes when a series of prisms is placed before the eyes to change the convergence required can be obtained from the graph on which the measurements are plotted. In this graph (fig. 11) the prismatic deviation base out is

plotted to the right (for then the convergence demand has been increased), and the prismatic deviation base in is plotted to the left. Above, on the ordinate, the esodeviation is plotted in minutes of arc, and below, the exodeviation. The data for this graph were obtained for an observation distance of 33 cm. As the base in prismatic deviation is increased, the disparity becomes increasingly an esodeviation, showing an esophoric oculomotor imbalance. Near the prism vergence limits the disparity

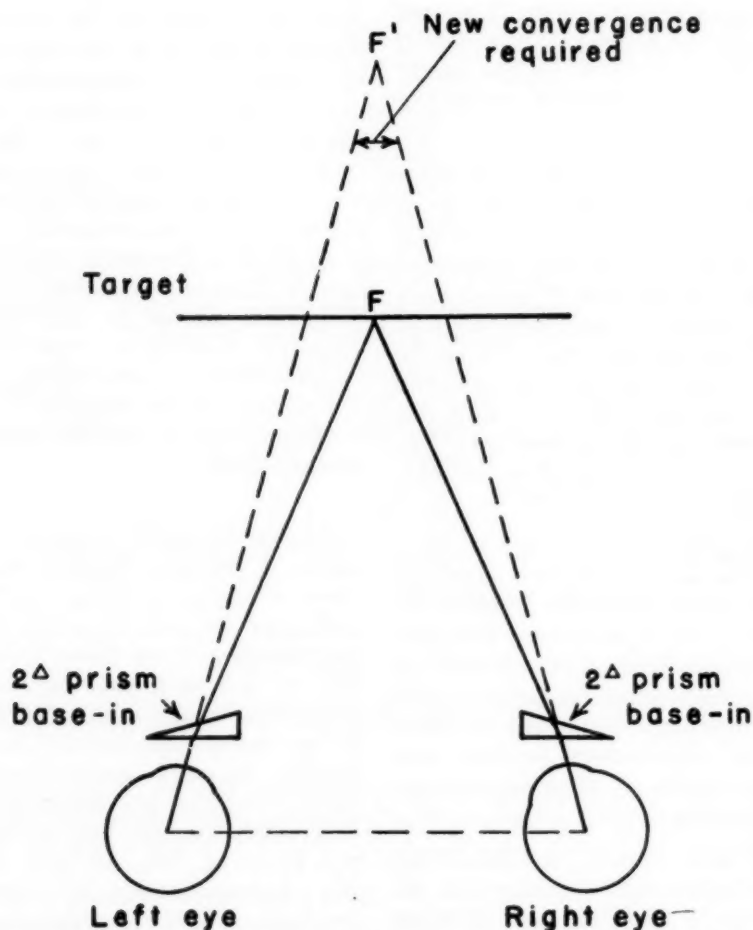


FIG. 10—Prisms placed base in before the two eyes, in causing a decreased required convergence for fusion, introduce a relative esophoria for an individual who is orthophoric for the observation distance represented by the point F.

becomes very large, and a further increase in prismatic power results in diplopia. The oculomotor imbalance is maximal at this point. The reverse occurs in the base out part of the graph where the exodeviation increases. Near the ends of the fusional amplitudes or prism vergences, the disparity then becomes very large. Since the subject whose data are illustrated was orthophoric, the curve drawn through the data points passes through the origin.

If the subject was exophoric for this

observation distance, a graph like that shown in figure 12 might be drawn. In this case, an exodeviation of nearly 21 minutes of arc is measured. As the base in prismatic deviation is increased, this disparity decreases and becomes zero for a prismatic deviation of 11.5 prism diopters. This point, P, at which the curve crosses the axis, is of special importance, for at this prismatic deviation the oculomotor imbalance is zero; therefore, this point corresponds to the equivalent phoria or the relative posi-

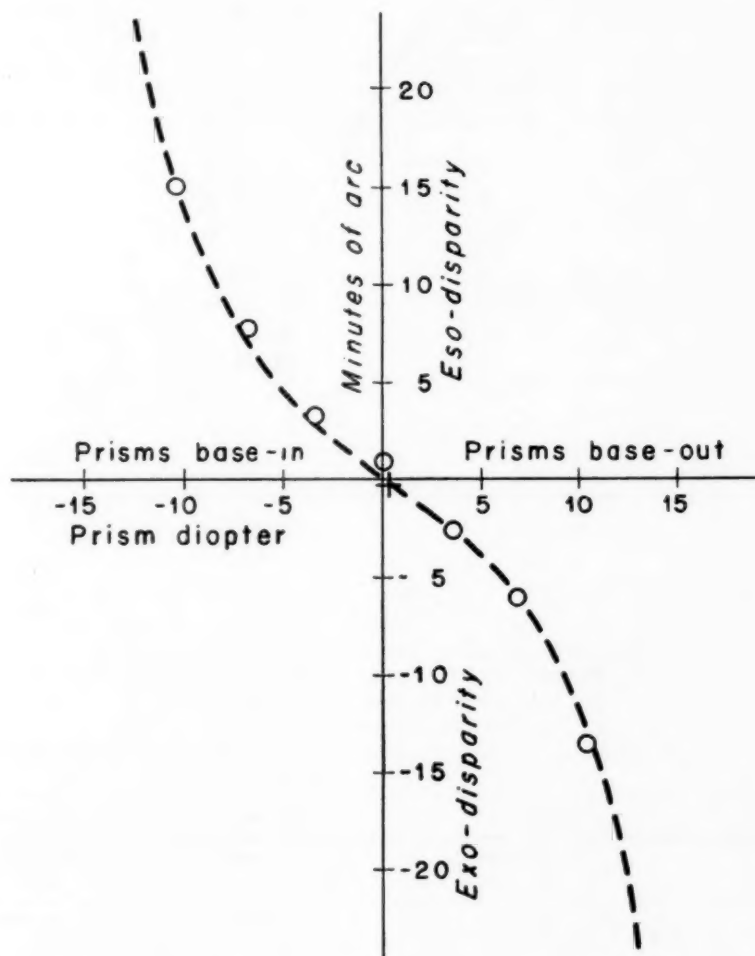


FIG. 11—Fixation disparity data for an orthophoric subject at the test distance, as prisms are placed base in and base out before the eyes.

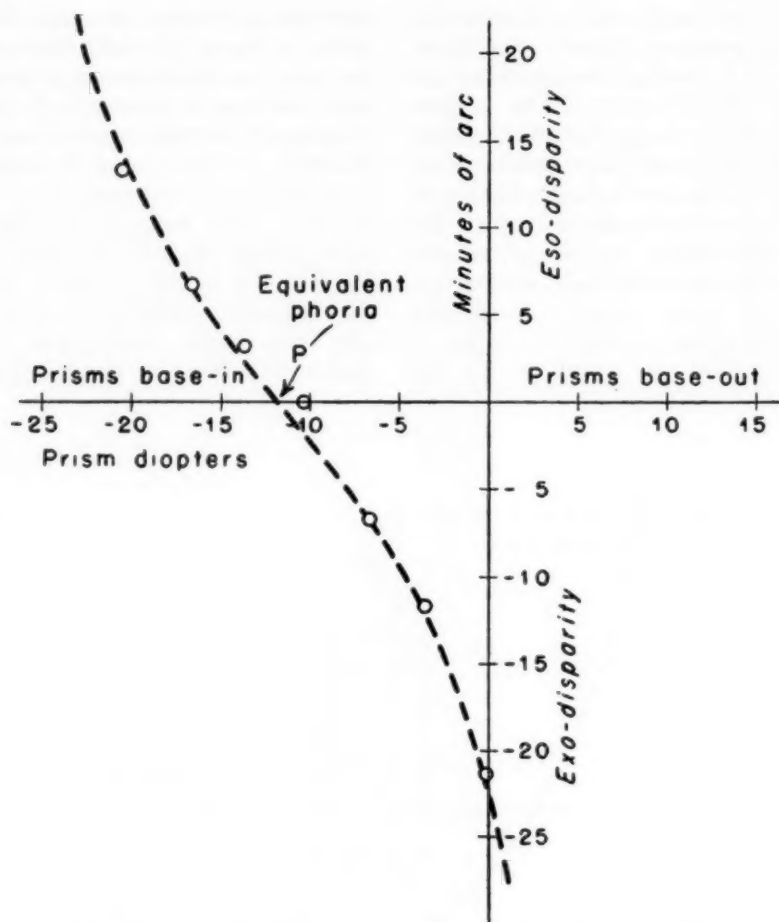


FIG. 12—Fixation disparity data for a subject who had an exophoria of 11.5 prism diopters at the test distance, as prisms are placed base in and base out before the eyes.

TABLE I
AN EXTREME EXAMPLE OF A STRIKING DIFFERENCE BETWEEN THE
PHORIAS MEASURED BY THE COVER AND MADDOX ROD TESTS AND
THE EQUIVALENT PHORIAS DETERMINED BY THE
FIXATION DISPARITY METHOD

OBSERVATION DISTANCE	METHOD		
	COVER	MADDOX ROD	FIXATION DISPARITY
Distant: 4.5 meters	6 Δ esophoria 3 Δ L.H.*	9 Δ esophoria 6 Δ L.H.	orthophoria 1 Δ R.H.
Near: 33 cm.	18 Δ esophoria 8 Δ L.H.	10 Δ esophoria 6 Δ L.H.	5 Δ exophoria† 1 Δ R.H.

*Left hyperphoria.

†With a reading add of 2.00.

tion of rest for the subject at this observation distance. As the base in prismatic deviation is further increased, the oculomotor imbalance becomes esophoric, and this esodeviation increases rapidly to the prism vergence limits. Base out prismatic deviation increases the exodeviation, rapidly approaching the prism vergence limit.

As a general rule, the equivalent phoria as measured by this technique agrees in a general way with Maddox rod measurements, although the latter tend to give a greater exophoria for near vision. There are many instances, however, in which the two are markedly different. An extreme example of a difference is shown in table I. This difference is so striking that it must be assumed that in many subjects fusion

modifies the innervational pattern to the extraocular muscles. Thus, the oculomotor imbalance measured by the equivalent phoria in the fixation disparity method in which fusion is maintained differs from that measured by the cover test, or by the Maddox rod test, in both of which binocular vision and fusion are prevented.

The actual magnitude of the fixation disparity is a quantitative measure of the oculomotor imbalance under the visual conditions of measurement and with the eyes at the required convergence for fusion. The actual magnitude of the phoria by itself does not tell the technician what the oculomotor imbalance is when the images are fused, although it is certainly related to that imbalance. This might be made clear

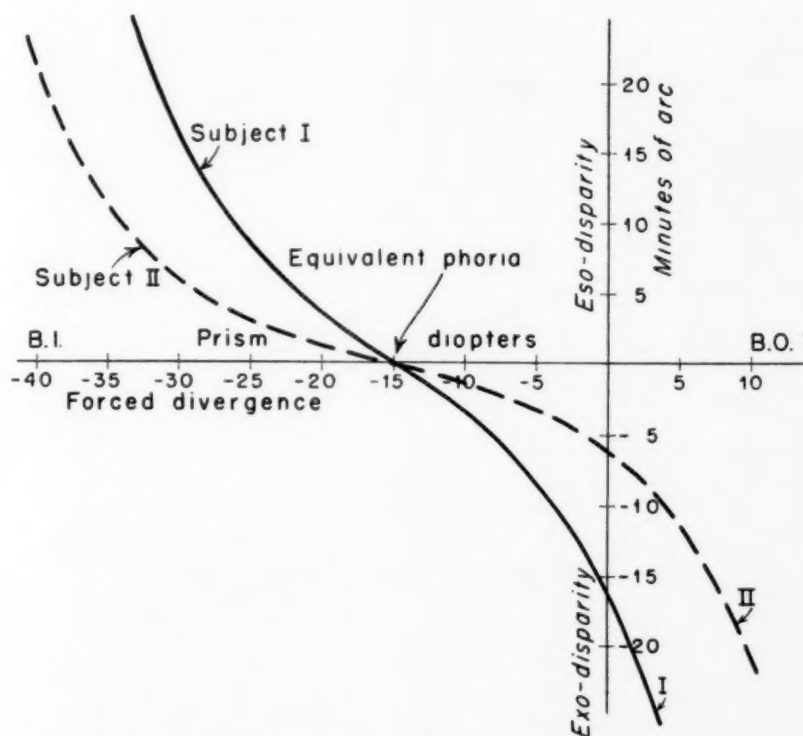


FIG. 13—Fixation disparity data for two subjects with the same exophoria but with different oculomotor imbalances.

from the idealized data of two subjects with the identical equivalent exophoria of 15 prism diopters (fig. 13). Though the equivalent phoria is the same for the normal conditions with the eyes at the required convergence for fusion (without any prisms), the fixation disparity of subject I is much greater than that of subject II. Accordingly, the oculomotor imbalance is greater in the first subject than in the second for two reasons: The rate of change of the imbalance with prisms is less in subject II, which may indicate that his compulsion for the fusion reflex is stronger than that of subject I. Secondly, the convergence required for fusion is much nearer the base out prism vergence limit for sub-

ject I than is that for subject II. Such data as these would bear on a certain body of general knowledge gained in clinical experience, namely, the difficulty that may be experienced in coping with an exophoria will depend not only on the magnitude of that phoria but also on the relationship between the convergence required for fusion and the base out prism vergences. The measurements of fixation disparity add another dimension to this problem.

The point is that the absolute magnitude of the fixation disparity is not a direct measure of the phoria. It is, however, a measure of the oculomotor imbalance under the ocular conditions of measurement. But, by the same token,

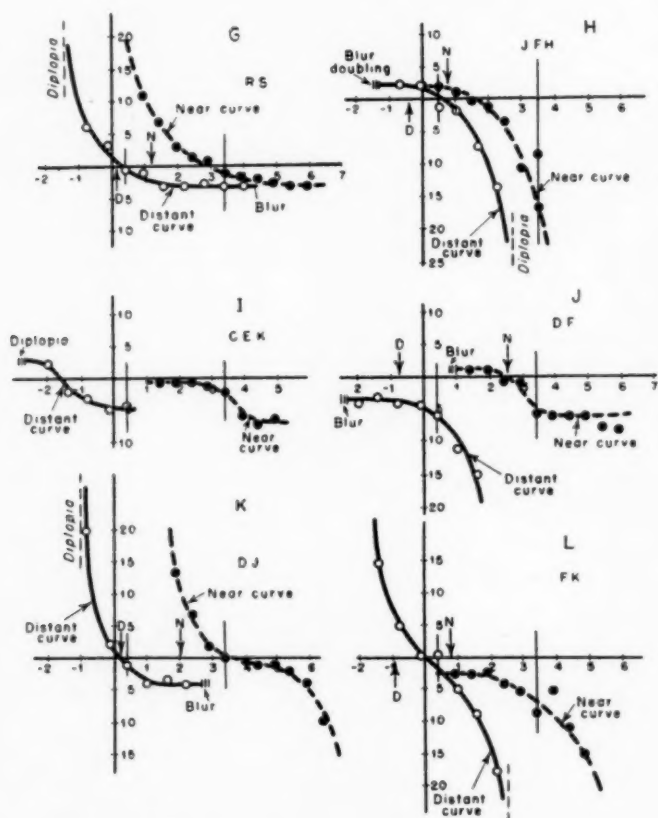


FIG. 14—Other types of fixation disparity-prism curves. These particular curves are plotted on an absolute convergence scale.

it can be argued that the phoria measurement alone is not a measure of the oculomotor imbalance when fusion is maintained at the required convergence.

The equivalent phorias obtained by this technique might be more thoroughly compared with the usual findings of phoria in patients who continue to have symptoms that could be attributable to motor imbalances.

The pattern of the fixation disparity curves obtained with prisms and with lenses could be studied more thoroughly in relation to clinical symptoms. In this discussion only the sigmoid type of curve has been illustrated, because it is this which is most often found. Three other types are found often, however (fig. 14). In the type shown in J and L of figure 14, the curve never crosses the abscissa, so that the equivalent lateral phoria cannot be found.

The measurement of the fixation disparity in the vertical meridian, as a measure of vertical motor imbalances, is also perfectly straightforward. The small nonius lines are then horizontal instead of vertical. In many respects the measurement of motor imbalances is simpler in the vertical meridian, since the accommodative and psychic convergences are not involved. There are also fewer deviations from the average pattern of response. Furthermore, the correlation of the equivalent vertical phoria with the hyperphoria measured by the Maddox rod is higher.¹³

It is not possible to go further into the complexity of the phenomenon at this time, but reference can be made to other papers on the subject. The importance of this phenomenon as a tool in the study of binocular motor behavior needs to be stressed, however.

The question may be asked: What is the practical importance of this to the orthoptic technicians or to the ophthalmologists concerned with problems of binocular motor behavior? Perhaps it would be more to the point to ask how this technique might be used in one's own work. At the outset it must be said that the final decision regarding the usefulness of this technique to clinical testing is yet to be decided. Certainly it is not a suitable tool for the study of that most important of patients, the young child, because of the need for accurate subjective responses. For special study or for general investigative work the technique may have much to offer.^{3,8,11,16}

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A SHORT METHOD FOR TREATING ANOMALOUS RETINAL CORRESPONDENCE

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NORMAL retinal correspondence is the most fundamental element of single binocular vision and is thought to be innate. This condition exists when corresponding retinal elements in the two eyes have a common visual direction. When heterotropia occurs, noncorresponding points are stimulated and certain adaptations ensue. This paper is concerned with the adaptation in which noncorresponding retinal points have assumed a common visual direction or, as it is commonly called, anomalous retinal correspondence.

For purposes of this paper, anomalous retinal correspondence may be divided into two types:

1. Unharmonious anomalous retinal correspondence — the angle of anomaly (the difference between the subjective and objective angles) is less than the angle of squint.
2. Harmonious anomalous retinal correspondence — the angle of anomaly is equal to the angle of squint.

If the angle of anomaly in either type is less than 5 prism diopters, the retinal correspondence is not considered to be anomalous.

We do not know why some patients with strabismus develop unharmonious and others develop harmonious anomalous retinal correspondence. The harmonious type usually occurs when a constant deviation is maintained over a long period of time; the unharmonious type probably occurs when the amount of deviation has changed.

Although there are several methods which may be used to diagnose anomalous retinal correspondence, the red glass diplopia test, the major amblyoscope, and the afterimage test give the necessary information.

The result of the red glass diplopia test depends on the position of the eyes in everyday life, since this position determines which retinal elements are being stimulated. If normal correspondence is present, the subjective and objective measurements will coincide. If the subjective and objective measurements are different, anomalous retinal correspondence is present. When suppression prevents diplopia, the use of a prism base up or base down before either eye will shift the image out of the suppression area, so that the subjective measurement may be taken.

If the subjective and objective angles are the same, or within 5 prism diopters of each other on the major amblyoscope, normal retinal correspondence is present. If the subjective and objective

I wish to acknowledge the assistance given by Miss Sally Moore.

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angles vary by more than 5 prism diopters, anomalous retinal correspondence is present. I am talking only about non-accommodative esotropia. There may be a variable shift on the major amblyoscope in accommodative esotropia which must not be confused with anomalous retinal correspondence. Since retinal correspondence may vary with targets of different sizes, the technician should use both peripheral and foveal targets when testing. In harmonious anomalous retinal correspondence, the subjective angle is at or within 5 prism diopters of zero, that is, the angle of anomaly is equal to the angle of squint. In unharmonious anomalous retinal correspondence the subjective angle falls between the objective angle and zero; in this case the angle of anomaly is less than the angle of squint.

TREATMENT

In the treatment of anomalous retinal correspondence, we have combined pre-existing methods with some ideas of our own. We have made use of occlusion, the major amblyoscope, the tracograph, physiologic diplopia, and prisms.

Occlusion

Total and constant occlusion is important in order to prevent stimulation of noncorresponding retinal points in daily life.

Major Amblyoscope

In treating anomalous retinal correspondence on the major amblyoscope, a target which subtends an angle greater than the subjective angle should not be used. Good fixation is a requisite to the following technique:

The slides used are simultaneous foveal perception targets (first degree), such as a pair having a 2.5 mm. dot on one slide, and a circle into which the dot exactly fits on the other slide. The dot is placed before the eye in which vision is suppressed and the amblyoscope arm is locked at the place on the

scale which measures one-half of the angle of squint. The slide containing the circle is placed before the dominant eye.

While constant fixation on the dot is maintained by the eye in which vision is usually suppressed, the patient is instructed to move the circle as close as possible to the right side of the ball. This is repeated, moving the circle from the left side to the right side of the ball. During this maneuver, the technician must observe carefully that the patient maintains constant fixation with the eye in which vision is usually suppressed. At first, the span of the arc may be as great as 40 prism diopters and, as the circle crosses, a previously unobserved hypertropia of as much as 10 prism diopters may be manifested. However, as the angle of suppression begins to break, the span of the arc will decrease toward the objective angle and the hypertropia will be reduced to zero. Because of suppression, either the dot or the circle will seem to disappear as superimposition is attempted, but the patient should be encouraged to continue this crossing movement until the dot is seen inside the circle as the circle moves from side to side. By the time superimposition is achieved, the subjective angle and the objective angle are the same.

In the event of harmonious anomalous retinal correspondence, it is not uncommon for the images to cross at the subjective angle for a period of time. If, after fifteen minutes, the objective angle has not been reached, the arms of the instrument should be placed so that the images fall inside the angle of squint. The orthoptist places her hand on the amblyoscope in such a position that the arm carrying the circle may not move behind the objective angle. The patient then moves the circle back and forth from inside the angle of

squint to the objective angle, always maintaining crossed diplopia. When, upon questioning, the patient reports that the pictures are coming closer together, the orthoptist will note that he does not wish to push the circle back into the subjective area. She then gradually moves her hand away and has the patient resume crossing the circle from the right side to the left side of the dot.

When simultaneous foveal perception is obtained with the dot inside the circle at the objective angle, foveal fusion (second degree) targets are placed in the machine and the patient is asked to move the pictures together. These pictures will usually be fused at his objective angle unless a slight amblyopia is present. When foveal fusion can be maintained for about one minute, larger fusional targets are placed in the cell behind the foveal targets, then the foveal targets are quickly removed. Since there are only three cells in the troposcope, these larger targets should be moved to the front cells, and this procedure repeated, gradually increasing the size of the fusional targets until the patient is able to maintain fusion with the largest picture that does not subtend an angle greater than his subjective angle.

After breaking foveal suppression, we begin developing vergence amplitudes, since the purpose of vergence amplitudes is to overcome disparate retinal stimulation in order to obtain bifoveal fixation. When there is a slight amblyopia present, bifoveal fusion is impossible. When a patient with an esotropia untreated by operation or a residual postoperative esotropia of no more than 8 to 10 prism diopters is treated for anomalous retinal correspondence, the deviation is frequently reduced into Panum's fusional area. Although bifoveal fusion is not present, disparate retinal stimulation within Panum's

fusional area gives rise to normal single binocular vision. If foveal suppression won't break and there is fixation disparity, the usual treatment for building up amplitude should be carried on.

Tracograph

Home exercises with the crossing technique of the ball and circle can be effectively carried out on the tracograph pictured in *Clinical Orthoptics* by Mary Kramer. The ball is placed so that the nondominant eye sees it in the mirror. A circle is pasted or drawn on a tongue depressor. The patient is instructed to place the circle on the table below the mirror so that the ball is seen



FIG. 1—Patient using tracograph for home exercises.

to the right. The circle is carried back and forth until superimposition has been achieved. The tracograph can be adjusted easily to the angle of squint, and since macular and foveal targets are used, we do not have to worry about the patient working in the subjective area too long. If a tracograph is not available, any local carpenter can make one (fig. 1).

In cases of unharmonious anomalous retinal correspondence, normal retinal

correspondence is usually obtained and stabilized on the major amblyoscope by the sixth to the ninth visit. Harmonious retinal correspondence requires more time for correction, and the patient may never develop normal retinal correspondence, especially if his squint is of long duration. When normal retinal correspondence has been obtained, or the anomalous retinal correspondence of harmonious type does not show signs of improvement by the ninth intensive treatment, surgical operation should be performed.

After the operation, the patient should be examined on the major amblyoscope to ascertain whether he can still maintain normal fusion. It is not uncommon for him to need two or three exercises on the major amblyoscope as a review. As soon as possible, however, exercises without the machine should be started. If there is a bothersome small tropia remaining following the operation, DFP is often useful in producing bifoveal fixation.

Physiologic Diplopia

We believe that physiologic diplopia is of prime importance in stabilizing normal fusion in daily life. Even though normal retinal correspondence was established and stabilized on the major amblyoscope, very frequently anomalous retinal correspondence is demonstrated on the red and green test. We have also observed paradoxical physiologic diplopia, or heteronymous physiologic diplopia, when it should be homonymous (figs. 2 and 3). To make it easier for the patient to recognize normal physiologic diplopia, we use a red filter in front of one eye, usually the right eye for convenience in remembering.

Let us consider a patient with postoperative esotropia. While he fixates a light 13 inches away, the technician covers and uncovers the right eye and asks him to report on which side of the near light the blinking red light appears. At first, he may report that the red and white lights change from one side to

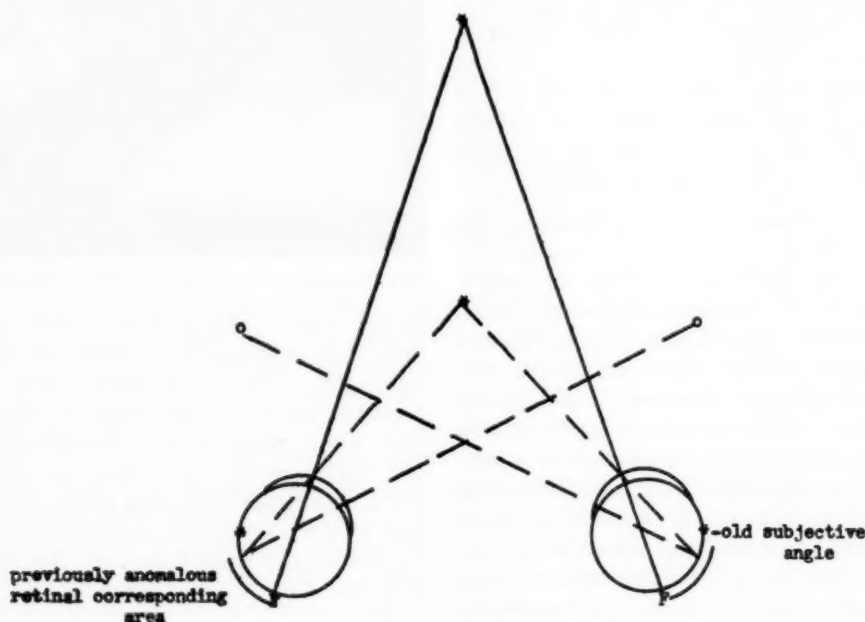


FIG. 2—Physiologic diplopia on distant fixation is important to establish normal fusion in patients with no postoperative exotropia. The area nasal to the old subjective area, including the fovea, has a temporal projection.

the other. The right eye is covered again to make him aware that the distant white light appears to the left of the near light. At about 5 second intervals, cover and uncover the right eye until he is able to see the distant red light flicker on the right side of the near light. Covering and uncovering

is gradually increased in frequency until the red light appears to stay on the right side of the near light. At this point, the covering and uncovering is stopped, and the patient is asked to observe that the near light appears in the middle between the red and white distant lights. It is important that the near

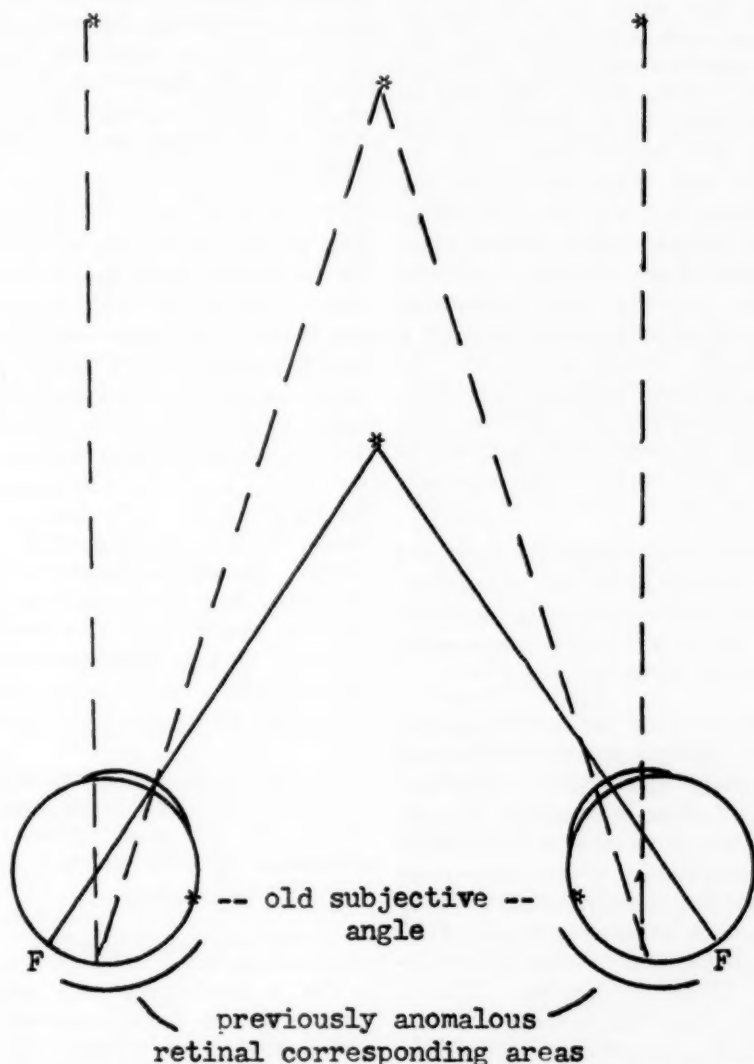


FIG. 3—Physiologic diplopia on near fixation is important for patients with no postoperative esotropia. The area which is temporal to the old subjective area, including the fovea, has a nasal projection, and projects paradoxically.

light be held straight in front of the patient's nose. If he is inclined to turn his head or to move the light to one side, he is not maintaining bifoveal fixation.

It is not enough that normal physiologic diplopia is present in the outer peripheral areas. Therefore, to establish normal physiologic diplopia from the peripheral areas all the way to the center, the patient must hold fixation on a near light while he walks toward the distant light, advancing until the distant images have emerged at Panum's fusional area at the foveas. He must be constantly aware of homonymous diplopia on the distant light. As he approaches the point at which the distant images fall into the old subjective area, he may again project paradoxical physiologic diplopia. At this point, the covering and uncovering technique must be repeated until he is able to regain normal projection. As an additional home exercise, the patient is given framing exercises, first on large objects and gradually working down to framing a dot which is seen bifoveally only.

A patient with surgically corrected exotropia would emphasize physiologic diplopia on distant fixation while walking back 20 feet, always maintaining heteronymous diplopia.

Once the patient has secured normal physiologic diplopia at both distance and near, absolute and relative vergences are begun. Relative vergence is even more important, since it is the relative vergence amplitudes which make clear single binocular vision possible. We try to obtain a relative divergence of 20 prism diopters and a relative convergence of 30 prism diopters. This is effected by prism base in and prism base out bar reading for near, and prism base in and base out on a Snellen vision chart at 20 feet with clear single bin-

ocular vision. Clear single binocular vision must be constantly maintained and, since letters are more easily memorized, we use a number chart. In order to make sure that there is no foveal suppression, constant supervision is required by the orthoptist. She should sit so that her face is directly in front of the patient's face and hold the target material so that the word being framed is directly in front of her nose. If bifoveal fixation is present, the bar will be in a straight line between the bridge of the patient's nose and the fixated word. If the slightest shift is observed as the patient's eyes reach the framed word in the center, bifoveal fixation has been lost.

To make it easier for the child, ask him to point to the letter which he sees in the middle between the frames and read it aloud; then ask him to point to the letters as he reads from left to right and from right to left, adding only one letter on each trip back and forth. This takes him into fusion between the frames where bifoveal fixation is present, and out of bifoveal fixation while he reads on the right and on the left sides. Each time he goes through the center, he obtains reinforcement, and gradually he can maintain straight eyes on the whole line. The procedure is repeated on lines with smaller print and so on until he can maintain clear single binocular vision on 20/30 print.

In the event that foveal suppression has redeveloped following surgical operation, we use a red filter over the dominant eye and use red and black print for bar reading.

PROGNOSIS

For a good prognosis, one cannot overemphasize the importance of early diagnosis and treatment. In infancy and early childhood, the binocular reflexes are in a state of diminishing flux

and capable of rapid destruction. If strabismus develops during this period, reflexes become abnormally conditioned and remain fixed throughout life unless treatment is instituted early.

Anomalous retinal correspondence develops only when eyes remain constantly heterotropic, and it becomes deeply rooted the longer the duration of squint. When surgical procedures are done very early on congenital heterotropia and the eyes are straightened, an opportunity is afforded for the normal reflex pattern to become established during the normal development period. Now, even though the patient no longer demonstrates anomalous retinal correspondence, only time and usage can securely establish the pattern of normal single binocular vision.

CASE REPORTS

Case 1

Girl, age six and a half years. Onset of exotropia at birth; it was constant at 20 feet and intermittent at near.

Refractive error:

O.D. = $-0.50 + 1.00 \times 90$

O.S. = $-0.50 + 1.00 \times 90$

VOD cc = 20/20

VOS cc = 20/20

XT cc = 20Δ (each eye fixing);

XT sc = 20Δ (each eye fixing)

X(T)'cc = 20Δ (each eye fixing);

XT'sc = 20Δ (each eye fixing)

Convergence near point:

Remote.

Red glass diplopia test:

At 20 feet—crossed diplopia of 5 prism diopters.

At 13 inches—alternate suppression.

Major amblyoscope test:

Subjective angle: Zero.

Objective angle: XT = 20Δ . Homonymous diplopia.

Afterimage test:

Positive—Normal retinal correspondence.

Negative—Anomalous retinal correspondence.

Treatment:

Six preoperative sessions of exercises were given over a period of three weeks. The patient came twice weekly. Before her operation she developed normal retinal correspondence according to all tests, and she had

an absolute prism convergence of 40 prism diopters for distance and 50 prism diopters for near. She had a relative convergence of 20 prism diopters for distance and 20 prism diopters for near. Physiologic diplopia was normal on both near and distance fixation. The positive and negative afterimages were normal. The near point of convergence was unlimited.

Surgical procedure:

Bilateral recession of lateral recti of 4.5 mm.

Four postoperative sessions of exercises were given with emphasis on the relative vergences.

She developed: relative divergence at distance of 10 prism diopters; relative convergence at distance of 25 prism diopters; relative divergence at near of 15 prism diopters; relative convergence at near of 30 prism diopters.

Case 2

Boy, first seen at age 7 years. Onset ET O.S. at age four and one-half years. He had one month of patching at age 5 years.

Refractive error:

O.D. = $+0.50S + 0.50 \times 90$

O.S. = $+0.50S + 0.50 \times 90$

VOD sc = 20/25+; with pinhole = 20/25+

VOS sc = 20/40; with pinhole = 20/30

ET sc = 30Δ ; ET cc = 25Δ

ET'sc = 35Δ ; ET'cc = 25Δ

Amount of deviation in cardinal positions of gaze:

Up: 35Δ ET'

Up Left: 30Δ ET'

Left: 40Δ ET'

Down Left: 45Δ ET'

Down: 45Δ ET'

Down Right: 45Δ ET'

Right: 40Δ ET'

Up Right: 30Δ ET'

Near point of convergence:

Unlimited. Vergence reflex good.

Punctum proximum of accommodation:

on a 20/30 symbol: 12 cm. D.D. and O.S.

Versions:

Essentially normal.

Red glass diplopia test:

Suppression O.S. distance and near. With vertical prism, homonymous diplopia of 4 prism diopters distance and near.

Major amblyoscope test:

Subjective angle = ET 5Δ .

Objective angle = ET $25-30\Delta$. Crossed diplopia.

Afterimage test:

Positive—Normal retinal correspondence; occasional anomalous retinal correspondence.

Negative—Anomalous retinal correspondence.

Treatment:

On the twelfth visit (seen twice weekly) he had developed normal retinal correspondence on the red glass diplopia test; on the positive afterimage test there was normal retinal correspondence; on the negative afterimage test there was normal retinal correspondence to anomalous retinal correspondence; on the major amblyoscope the objective and subjective angles were at 20-25 prism diopters base out. Normal fusion was demonstrated from the fovea to periphery. He diverged to 15 prism diopters base out and recovered at 18 diopters base out, and then converged to 35 diopters base out and recovered at 28 diopters base out. (Absolute vergences.)

VOD = 20/25

VOS = 20/25

Surgical Procedure:

Recession of both medial recti of 3.5 to 4 mm.

On the fifth postoperative visit, he showed

VOD = 20/20; VOS = 20/20; esophoria sc on 20/25 letter = 6.

Amount of deviation in cardinal positions of gaze:

Up: 6 Δ E'

Up Left: 1 Δ X'

Left: 2 Δ X'

Down Left: 5 Δ X'

Down: 8 Δ E'

Down Right: 4 Δ X'

Right: 4 Δ X'

Up Right: 2 Δ X'

Versions:

Normal.

Near point of convergence:

1 inch—fusion reflex excellent.

Red glass diplopia test:

Fusion at distance and near.

Absolute prism divergence at distance = 5 Δ ; convergence at distance = 20 Δ .

Absolute prism divergence at near = 8 Δ ; convergence at near = 20 Δ .

Relative divergence at distance = 10 Δ ; convergence at distance = 20 Δ .

Relative divergence at near = 25 Δ ; convergence at near = 20 Δ .

Major amblyoscope:

Esophoria = 3 Δ (objective and subjective angles).

Good foveal fusion.

Diverged to 5 Δ base in and recovered at 0.

Converged to 25 Δ base out and recovered at 15 Δ base out.

Afterimage test:

Positive—Normal retinal correspondence.

Negative—Normal retinal correspondence.

Panel Discussion: Occlusion

I

TEMPORAL OCCLUSION IN CONCOMITANT CONVERGENT STRABISMUS

ANGELA SWENSON

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FOUR years have passed since the paper "Temporal Occlusion in Concomitant Convergent Strabismus" was first printed in *The American Orthoptic Journal* (3:48-50, 1953). Temporal occlusion was first used only in cases of eccentric fixation. It is now used for patients with anomalous retinal correspondence and central fixation and occasionally for patients with normal retinal correspondence, principally for the patient with strabismus and poor accommodation. Temporal occlusion is a good postoperative stabilizer. It is not a cure-all; it does not replace any of the usual instrument techniques, but it does supplement them. I feel that the Walraven technique should be postponed at least until localization is true.

The patch is no longer cone-shaped, since only a temporal blocking of the optical lens is necessary. This patch is applied quickly with gummed postage tape on the optical lenses. If the lenses are plastic, masking tape is used. Masking tape is not as satisfactory because it can be altered more easily by the child. With the good eye fixating on a light held 30 cm. from the patient in a midline position, the lens before the deviating eye is marked at the center of the pupil and the patch applied temporally to this mark.

First grade targets are used to plot the suppression area by noting the anomalous crossing points. Most young children are very cooperative and like the challenge of trying to put the lion in the cage and a car in a garage, etc. When the deviation is over 18 prism diopters from objective, the first grade targets cannot be superimposed but will just cross over when the suppression area is by-passed. When the deviation is under 18 prism diopters, the patient will often claim superimposition at an angle lower than the objective angle. On close questioning the patient will admit that the target before the dominant eye is straight ahead, and that the other target is merely assumed to be straight ahead but is not actually seen. When the deviation is less than 10 prism diopters, he may or may not be able to see the first grade targets actually superimposed at a lesser angle than the objective angle. When the patch is worn faithfully, this suppression area reduces. The length of time varies depending on the age of the patient at the onset of deviation (whether the deviation was present at birth or acquired), on his faithfulness in wearing the patch, and also on whether it was correctly applied. The line of demarcation must be at midpoint of the pupil of the deviating eye with the dominant eye fixating on the light in the midline position. This is the cause of some of the misunderstanding per-

Presented at the Midwestern Regional Meeting of the American Association of Orthoptic Technicians, May 13-14, 1957, Madison, Wis.

taining to the application of the temporal patch.

The patient's glasses are usually at just the correct distance from the eyes for this type of occlusion. When the patient has deep-set eyes and a high nasal bridge, the glasses are so far from the eyes that the line of demarkation does not block the retinal stimulus at the proper point. Holding a piece of cardboard in front of a flashlight will give the same response in the reflection on a wall. The farther the cardboard is from the light, the less occlusion or shadow appears on the wall. Glasses too close to the eyes are unsatisfactory also.

The parent usually thinks the temporal patch will create a greater convergent deviation. This would be the tendency if the patient has normal correspondence because it actually is a convergent stimulus if the objective angle is at 0 or on the convergent side. The unpatched portion of the lens is the most important in partial occlusion. In some cases of normal correspondence the temporal patch may be annoying and confusing. In anomalous retinal correspondence the temporal occlusion is a relief; it gives the patient an opportunity to relax, and his deviating eye then relaxes under the patch. When the area of suppression is under 10 prism diopters from the objective angle on the troposcope, total occlusion of the dominant eye can be started. Retinal rivalry is sometimes noticed at this stage. The objective and subjective angles are at the same point, but the eye is actively straightening or converging, so that the light reflex is either at the proper point or decentered. The technician must be very observant at this stage and always alert to follow the objective angle. This is a very confusing statement and has to be demonstrated. Action has to be seen to be appreciated. It is very similar to observing a patient hold fusion toward orthophoria and

then noticing a breaking and recovery action but it is slightly different in character.

As stated in the previous paper, in some cases, the suppression area with the anomalous crossings seems to increase again following full occlusion of the dominant eye. When this occurs, it is wise to resume temporal occlusion. Peeking may be largely responsible for this. Patients with alternating squint do not have a visual problem, but they do have a deep suppression area and wide anomalous crossings.

Temporal occlusion might eventually make fusion possible in all healthy eccentrically fixating eyes if the patch is worn faithfully. The length of time for which the patch must be worn will depend upon (1) age of the patient when first applying the patch, and (2) the age at onset or the duration of deviation. For instance, if the patient is 6 years old and the deviation was present at birth, correction would require that the patch be worn for approximately three years. If the patient has active orthoptic training at least once a week, this time can be shortened considerably. The duration of wearing the patch would change proportionately with the age at application and the age at onset.

The dominant eye of the amblyopic patient with normal retinal correspondence is totally occluded from the beginning. Most of these patients accept the total occlusion without much protest and have very little psychologic or emotional upset. However, in some patients, the improvement in visual acuity eventually seems to stop. A brief session of temporal, or alternate temporal and total, occlusion will sometimes give the needed boost. Sometimes a slight diagonal angle of the patch will change or alleviate a hyperphoric or cyclophoric action. When doing accommodation studies on older patients, and sometimes on the younger children, with a near E chart, the studies show

more rapid accommodative improvement after temporal occlusion, especially in the low angle deviation. The reason for this is that the temporal occlusion permits binocular vision at the near point, whereas total occlusion completely dissociates the accommodation-convergence relationship.

SUMMARY

After five years, temporal occlusion is still considered a valuable part of therapy for those difficult patients showing eccentric fixation or a deep suppression area with anomalous crossings. Temporal occlusion produces the following changes:

1. A change in the character of fixation in the amblyopic eye

2. A decrease in the angle of squint and, in some patients, orthophoria
3. An increase in the visual acuity of the amblyopic eye
4. An improvement in accommodation in low angle deviations

Temporal occlusion is always applied to the lens before the convergently deviating eye; total occlusion is always applied to the dominant eye.

Eccentric fixation and anomalous correspondence are the counteraction of the deviation. Temporal occlusion does not primarily counteract the deviation; rather, it counteracts the counteraction.

II

NASAL OCCLUSION

ANITA J. STELZER

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NASAL occlusion is equally as effective in eliminating suppression for divergent deviations as temporal occlusion is for convergent deviations. The technique of applying the occlusion is the same as that for patients with convergent squint, except that the shielding material is applied to the nasal half of the lens, covering half of the pupil. The suppression areas can also be plotted by finding the crossing from heteronymous to homonymous diplopia and then approaching the area from the homonymous side toward the heteronymous to find the transfer point from one type of diplopia to the other.

Patients with exotropia with the greater deviation for distance, or patients with excess divergence, are benefited particularly by nasal occlusion. Measuring the amount of occlusion for these youngsters is more difficult, since it must be measured when a distant object is fixated with the dominant eye. The deviating eye is occluded. These youngsters, however, are the ones who are helped particularly by this type of occlusion. The suppression by patients with divergence excess is much deeper than that of patients with convergence insufficiency. We have found it very important to eliminate all the suppression

between orthophoria and the objective angle as the first step toward developing good stable fusion and in the maintenance of constant parallelism and binocular vision.

A study of 138 patients with divergent strabismus was made using patients referred to the St. Louis Ophthalmic Laboratory over a period of two years who received active training during some part of that time. One-half the patients had hypermetropia and less than one-third of the entire group had myopia. A hypermetropic error or cor-

pression accompanying divergent deviations is less difficult to overcome.

I prefer to overcome suppression in both esotropia and exotropia before surgical correction is made and the deviation and the suppression area are altered. Referring ophthalmologists have supported this theory so I have been able to work with many patients.

The amount of deviation in the tropias of these patients is usually above 20 prism diopters of exotropia and suppression is quite deep. Relative locali-

TABLE I
RESULTS OF ORTHOPTIC TRAINING IN 138 PATIENTS WITH DIVERGENT STRABISMUS

TYPE	NO.	TOTAL RESULT		HYPERMETROPIA		MYOPIA		NO ERROR	
		GOOD	POOR	GOOD	POOR	GOOD	POOR	GOOD	POOR
Concomitant divergent strabismus	39	29	10	14	5	5	1	10	4
Noncomitant divergent strabismus	29	17	12	9	8	6	4	0	0
Convergence insufficiency	39	31	8	17	3	5	0	9	5
Divergence excess	31	22	9	10	1	5	0	7	8

rection is certainly not conducive to improvement or strengthening of weak convergence. One of these patients had surgical treatment before orthoptic training and six had surgical treatment during training. All of these were among those who were rated in the poorer results (table I).

While it is generally believed that anomalous retinal correspondence is seen less frequently in patients with divergent deviations, we have treated many patients with quite large divergent deviations, and these apparently have an area of suppression similar to that seen in patients with convergent deviations. In most cases, however, the sup-

pression accompanying divergent deviations is less difficult to overcome.

By applying nasal occlusion on the deviating eye, and a red kodoloid over the dominant eye, the patient can be taught to fixate first the red light and then the white light. When doing this he almost instantly learns to localize the images properly. The first requisites for good results are proper turning of the head to institute macular fixation of the deviating eye at all times and proper, constant wearing of the glasses or frames. Without these requisites half lens occlusion is useless.

The occlusion is removed for troposcope work, but the effect of its proper use at home is carried over into that area. I use the Walraven technique in all my training. The more interesting picture is placed before the deviating eye and steady fixation is maintained with that eye. This in itself stimulates a dormant and suppressing macula. After the objective angle is determined and the end goal is established, the technician places the images in a heteronymous position (maintaining fixation with the deviating eye) even if that position is at 30 prism diopters base out. If and when this proper diplopia is not spontaneously present, the technician uses other means of stimulation such as either lateral or vertical movement of the nonfixed image. When heteronymous diplopia is spontaneously seen and recognized, the images are moved toward each other with the deviating eye still maintaining fixation in an effort to hold that position until the objective angle is attained. When superimposition with first grade targets can be maintained steadily at the objective angle without even fleeting suppression, the patient begins trying to develop fusional amplitude. It is rather astounding how easily the fusional amplitude develops in most instances when the problem is approached in this way.

During this time the children are also being trained daily at home. Since mothers are so busy and children so easily bored with homework, I utilize television for this. For at least two programs each day, they are asked to use the red kodaloid over the dominant eye with the nasal occlusion in place and are then asked to find the red and white screen. After they have learned to localize the images, they should watch the white screen constantly. The tech-

nician stresses the consciousness of two screens and their proper relationship in space. Later the patient is given fusion homework in which the mother must do her part and work with the child to interpret fusion and diplopia and to develop the fusional amplitude.

When amblyopia occurs in patients with exotropia I do not hesitate to use full or total occlusion. Amblyopia requires full occlusion only for very few patients—when anisometropia was a precipitating factor. The criteria for really good results are:

1. Loose prisms
 - A. Distance—convergence to 50 prism diopters base out, relaxation to cover test deviation.
 - B. Near—convergence to 50 prism diopters base out, relaxation to cover test deviation.
2. Troposcope
 - A. Convergence to 60 prism diopters base out.
 - B. Relaxation to objective deviation.
3. Advanced homework with stereopsis (*Miss Eyles' Functional Home Exercises*)
4. Keep eyes straight at all times
(Ability to read visual acuity chart binocularly with eyes straight)

In checking the length of time that the nasal occlusion was worn, I found that technicians were very reluctant to remove it and that the fusional amplitude is almost at its goal when the technician will permit removal, at first indoors, under observation, and later all day except on snowy or brilliantly sunny days. The parents should get the youngsters quite dark sun glasses as an added protection when the nasal occlusion is no longer necessary.

III

THE USE OF TOTAL AND CONSTANT OCCLUSION

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ONE of the most serious sequels of strabismus is poor vision in the crossed eye. The ophthalmologist's first concern, following a thorough refraction, is the improvement of the visual acuity in the amblyopic eye. If the amblyopia becomes deeply rooted, the visual acuity will be greatly reduced and eccentric fixation may result. Amblyopia can and should be remedied in early childhood or as soon as it is known to exist. To effect an appreciable improvement in visual acuity with existing means of treatment once adult life is reached is practically impossible.

Total, constant, and complete occlusion of the nonamblyopic eye is the most effective method of treatment and should be started as soon as the amblyopia has been discovered. The improvement of visual acuity in the amblyopic eye will usually be rapid if the occlusion is carried out correctly and the cooperation of both parents and patient is good. The rapidity of improvement and the prospect for a permanent result will decrease after the age of 7 or 8 years and will become increasingly slower as the child becomes older.

There are some cases of amblyopia which will not improve, even though the occlusion has been carried out for a prolonged period of time. This condition is described as intractable amblyopia.

In other cases of amblyopia, the visual acuity will improve until a certain level is reached. Since visual acuity normally develops during the first five years after birth, onset of a deviation during this period may arrest the development at

the stage which it has reached. This stage seems to be the level beyond which such cases cannot improve even though an occlusion program is followed faithfully.

Factors influencing the effect, duration, and success of the occlusion are:

1. Age at onset of squint
2. Age when occlusion is begun
3. Visual acuity and fixation when occlusion is begun
4. Cooperation of patient and parents

In our records, there are many cases where adequate total occlusion has yielded good results; however, there are few where total occlusion of the same eye has been used alone during the entire course of equalization of visual acuity. Any current evaluation is likely to elicit other factors which require attention (e.g., presence of a vertical deviation, change of angle with new glasses, change of preference, or visual demands for school work which cannot be ignored). Consequently, another form of occlusion may be prescribed at that time. Although the end result is still gratifying, it has been the culmination of combining one or more other forms of occlusion with total occlusion of the originally dominant eye. This is, of course, in accordance with the subject of this paper.

To be truly successful, occlusion should be constant and total until the visual acuity of both eyes is equal. This point cannot be stressed too highly. Wearing the occlusion just a few hours during the day will not only prolong the

treatment but is unpleasant for the child, and it may have an adverse effect on the nervous system. When occlusion is first prescribed, the visual acuity of the amblyopic eye should be checked at regular intervals by the ophthalmologist and by the orthoptist. The parents, as well as the child, will need encouragement, especially if this is their first experience with occlusion.

The question as to how long the non-amblyopic eye should be occluded cannot be answered in absolute figures because it varies with each patient. The general agreement is that the better eye should be occluded until the visual acuity in the amblyopic eye has been brought up to normal or at least to the level of the better eye. If the visual acuity has not improved after a period of three to four months, the likelihood that it will improve at all is slim. But there have been cases in which the visual acuity did not improve until the occlusion had been carried out for six months or more. For the most part, the speed of improvement depends upon how deeply rooted the amblyopia has become. If the child is severely handicapped by the occlusion and becomes extremely upset, it would be wise to discontinue the occlusion or substitute some other form of treatment.

Total occlusion may be used for conditions other than amblyopia. The following are examples:

1. To improve motility, especially abduction
2. To overcome or prevent anomalous retinal correspondence
3. To overcome suppression
4. To relieve constant diplopia or confusion
5. To maintain the eyes in a position favorable to the correction of a residual angle of deviation after an operation for squint which has not been entirely successful

6. As an aid to the investigation of of a case of heterophoria (Marlow's occlusion) and occasionally to overcome a suspected amblyopia associated with heterophoria
7. To teach alternation by alternate occlusion
8. To break up or prevent undesirable habits by occluding the eccentrically fixating eye as a preliminary step to temporal occlusion of that eye

The practice of occluding the eye which fixates eccentrically is a comparatively new procedure and not generally used as a part of the treatment of eccentric fixation. We have used it approximately a year at the suggestion and under the direction of some of our ophthalmologists. Although this is too short a period from which to draw any specific conclusions, we have been encouraged by promising signs and found that the visual acuity of the amblyopic eye has not retrogressed. The total occlusion of the amblyopic eye will give the eye a complete rest from any sort of fixation. The fixation, in such a case, may be of a wandering type at the next visit. There may actually have been some improvement in visual acuity. Of course this is not true in all cases. Time will tell whether or not this preliminary procedure will have any value.

Total occlusion may be substituted, and quite successfully in some cases, for other types of occlusion. For instance, it may be substituted for nasal occlusion to teach alternate fixation and overcome suppression. The total occlusion is carried out alternately, or on the non-suppressing eye, and should be used for patients whose visual acuity is fairly equal and who have the nonaccommodative type of squint. This procedure of teaching alternate fixation and overcoming suppression is probably not the most successful or popular method, but it can

be used. (I mention it mainly as a possibility.)

Other forms of occlusion have been used for eccentric fixation, since the practice of totally occluding only the eye which fixates centrally is useless or, as thought by some, actually harmful. One of the forms of occlusion used is the temporal occlusion of the amblyopic eye, developed by Miss Swenson and Miss Stelzer. Another consists of totally occluding the eye which fixates eccentrically instead of the eye which fixates centrally in order to break up the habit of eccentric fixation. Temporal and total occlusion recently have been combined in order to enhance a promising prognosis. The most common combination is the temporal occlusion of the deviated eye until the fixation has become wandering instead of eccentric, then proceeding with the total occlusion of the eye which fixates centrally.

From recent studies of different types and combinations of occlusion in eccentric fixation cases certain conclusions have been evolved. One cannot state with certainty that one type of occlusion is more successful than another in eccentric fixation, since there are too many controlling factors influencing the result of the treatment. With each individual case the factors vary. Occlusion of the better eye is not contraindicated, although the cooperation of the patient is harder to obtain in this type of treatment. Better results can be expected if temporal or complete occlusion of the eccentrically fixating eye is followed by surgical straightening of the amblyopic eye after central fixation has been obtained.

Summarizing the different combinations of total and temporal occlusion, we find three main types with variations in each type, the variations depending on the progress of the patient:

1. Temporal occlusion of the eye which fixates eccentrically followed by total occlusion of the eye which fixates centrally as soon as the fixation begins to wander in the amblyopic eye
2. Total occlusion of the eye which fixates eccentrically, followed by temporal occlusion of the same eye and then total occlusion of the nonamblyopic eye
3. Total occlusion of the eye which fixates eccentrically until the fixation wavers, then total occlusion of the nonamblyopic eye

When total and constant occlusion has been prescribed by the ophthalmologist, the orthoptist is usually responsible for instructing the parents and the patient how to make and correctly use the occlusion. During this initial instruction period, the orthoptist must be certain that either or both the parents, as well as the patient (depending on the patient's age), understand the instructions fully. This is also an opportune time for the orthoptist to clarify for the parents and patient any points the doctor has made, or to answer any questions concerning the purposes of occlusion or the degree of amblyopia.

We have found that the Elastoplast* occluder works best for us in total, constant occlusion because it allows the patient to keep his eye open while he is wearing it. This patch also provides ventilation. If made and worn correctly, it will eliminate peeking and many of the complaints which accompany constant and total occlusion.

The instructions for making the Elastoplast occluder (fig. 1) are as follows: From a roll of Elastoplast or Elastikon**

*Elastoplast is manufactured by Duke Laboratories.

**Elastikon is manufactured by Johnson & Johnson.

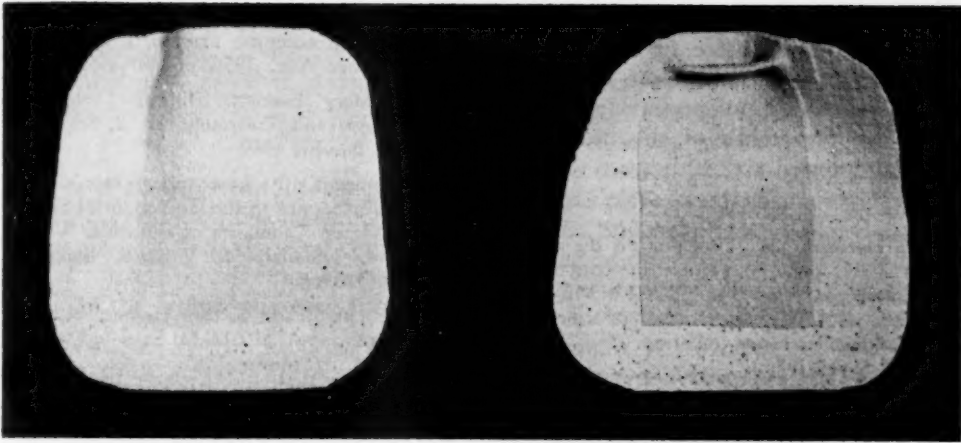


FIG. 1—The Elastoplast occluder for total, constant occlusion. It provides ventilation but eliminates peeking. (Left) Face down. (Right) Face up.

3 inches wide, cut two pieces, one 3 inches wide and the other 1 inch wide. Trim the corners of the large (3x3) square and place the 1-inch strip in the center of the square to within one-half inch of the end (with face down so that the tape will not stick to the eyelid). Cut off one-fourth inch of the one-half

inch residue and reattach this piece to each side of the 1-inch strip at the top of the patch, forming a space which provides for ventilation. Fold the small strip upon itself right up to the edge of the 1-inch strip on both sides.

The size of the patch will depend upon the size of the patient's face. For a very young child the patch may have to be made smaller than 3 inches, and accordingly, a larger patch may have to be made for an adult.

Place the patch on the eye so that the inner edge follows the center of the nose and the bottom edge is adjacent to the outer edge of the nostril. The vent (open space) should point upward and outward enough to avoid crowding the glasses. If glasses are worn, the occluder is worn *under* the glasses to prevent peeking (fig. 2).

The Elastoplast should be kept in a container with a tight lid. If it becomes too dry, a moist piece of cotton or tissue may be placed in the container with it.

It has not been my intention to discuss fully the various types of occlusion, but, instead, to review a few of the most common types and to emphasize a few



FIG. 2—Patient wearing occluder with the air vent pointed upward. Notice the occluder is worn under the glasses to ensure complete occlusion.

points concerning their use in orthoptic treatment. The choice of the type of occlusion to be used depends, naturally, upon the condition present and also upon the preference of the individual ophthalmologist and orthoptist.

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IV DISCUSSION

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TEMPORAL occlusion is a brilliant and ingenious solution to the problem of eccentric fixation and anomalous projection in esotropia. Temporal occlusion eliminates some of the offending stimuli to the retina of the deviating eye, thus preventing any reinforcement of anomalous projection and encouraging a more correct orientation of the deviating eye. The results obtained by Miss Swenson and others leave no doubt as to the efficacy of this type of occlusion in appropriate cases.

Half occlusion, temporal or nasal, has still another useful application. We sometimes encounter patients of school age with fairly deep amblyopia and normal correspondence who would be candidates for full occlusion of the fixing eye except that this would interfere with school work. Half occlusion during the school year, while less than ideal, is better than postponing any occlusion until summer vacation.

Nasal occlusion is a logical outgrowth of temporal occlusion; the same theoretical considerations apply. We are indebted to Miss Stelzer for her demonstration that half occlusion is also of value in appropriate cases of exotropia.

Her observations on the distribution of refractive errors in exotropia are generally in agreement with recent published studies and lend further weight to the growing conviction that refractive errors have little influence on most cases of exotropia.

It is encouraging to learn that Miss Stelzer has worked out a plan of training which can be followed in the home but doesn't require that the mother herself be an amateur orthoptist.

Mrs. Swift has mentioned something that is probably new to most of us; namely, the total occlusion of the deviating eye in cases of eccentric fixation. At first this sounds paradoxical, but the principle is analogous to that of tem-

poral or nasal occlusion. The idea makes sense, but, as Mrs. Swift has said, time will tell whether it has clinical value.

There is no doubt of the value of total, constant and complete occlusion when it is indicated. Any compromise usually leads to difficulty and serves to prolong the length of treatment.

One more form of occlusion which is sometimes helpful is the use of clear nail lacquer on the front of the spectacle lens before the eye which habitually fixates. Full occlusion may succeed in improving the vision in the squinting eye to the optimum, but the patient continues to fixate monocularly, instead of alternating freely, once the occluder is removed. Blurring the vision of the fixating eye by painting the lens of the patient's glasses with nail lacquer gives him a free choice of which eye to use, but experience teaches him that he can see better with the deviating eye. This helps the patient learn awareness and control of which eye is fixating, and often leads to free alternation after the lacquer has been removed. Acetone is a good solvent for the lacquer.

So far we have confined our discussion to concomitant deviations. In non-comitant heterotropia in which there is a definite paresis of a specific muscle, the same principles of occlusion are not necessarily applicable. Occlusion of part of a lens is sometimes used to prevent fixation with the paretic eye in the field of action of the paretic muscle, in order to prevent reinforcement of a secondary deviation. If there is amblyopia, total occlusion of the sound eye is of course indicated for the improvement of vision, but there may be an increase in the angle of squint owing to the development of secondary deviation.

Finally, I want to emphasize what Mrs. Swift has already stated so very well: "Amblyopia can and should be remedied. . . as soon as it is known to exist." The best treatment of amblyopia and eccentric fixation and anomalous retinal correspondence is to prevent them. Supervised occlusion should be started as soon as a squint becomes constant or almost constant. It isn't necessary to test the vision to know that amblyopia is developing. We should anticipate amblyopia and anomalous correspondence before they get a chance to develop. We may not know the fundamental pathologic physiology of amblyopia but we do know the precipitating cause and we've known it for a long time.

All this, of course, is obvious to every one of you. If it were understood by the general public there would be no need for us to have this panel discussion on the distressing problems which result from neglect. Our problem is not so much one of knowing what to do as it is one of educating the public to seek treatment when strabismus first begins to develop.

Unfortunately the idea persists that a child might outgrow a squint. Too many parents are concerned only with the appearance of strabismus and are unaware of the visual complications which can and do develop. There is a need for more education of the public regarding amblyopia.

The orthoptic exhibits at pediatric conventions are a most welcome step in this direction. However, pediatricians don't take care of all children, and parents don't always consult their doctor right away about matters that aren't especially urgent. But many do consult Dr. Spock's book on child care. It is the

mother's bible. Millions of copies have been sold and most of them are dog-eared from repeated usage. None of us would quarrel with what Dr. Spock has to say about "crossed eyes," but we might recommend that he add a concise few lines about the development of amblyopia and other complications in neglected cases. Dr. Spock is now in the Department of Pediatrics at Western Reserve University, Cleveland, Ohio. I hope that some of you will feel inspired to make your opinions known to him.

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DEVELOPMENT OF MACULAR VISION

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ACHIEVEMENT of perfect binocular, single, macular vision is the goal in every refraction, cataract operation, corneal transplant, operation to correct squint, and orthoptic procedure. In cases of deviations of the visual axes resulting in manifest strabismus, the results of these procedures are all too often disappointing. The question of how long the technician and physician should continue the effort is one that raises many differing opinions and even, at times, heated argument.

Let us review the anatomical development of the central retinal area. At about the third month of intrauterine life, the macular region lags behind the rest of the retina. At the seventh month it begins to catch up, and there is accelerated growth and differentiation of this area. At term, the organ is not yet fully developed, and evidence indicates that the perception of light may play a part in the final stages of formation of the adult macula and fovea.

No rods are present in the depths of the fovea centralis, and the cones are more numerous in this area than they are in the peripheral retina. The cones are not only more numerous, but they are modified in shape, actually resembling rods. The increase in the number of cones is probably the reason that we are able to see in such great detail

when we direct our gaze in such a way that the image falls on the fovea. Nature has arranged the retina in this spot so that there is little tissue for the image to traverse to get directly to the receptor cells.

This function of detailed vision, however, is not ready for action until about the fourth month of postnatal life. In the interim there is often some aimless deviation of the eyes that is sometimes mistaken for squint. Any such deviation should be observed carefully, for it may be the start of manifest strabismus. However, in most instances when the maculas are fully developed the eyes align themselves in the normal manner. Any deviation after that period can be considered pathological.

Deviation of the visual axes does not always involve defective macular vision, nor does the defect of macular vision known as amblyopia ex anopsia always accompany deviation of the visual axes. No one theory is accepted as to the cause of this amblyopia. My own impression is that heredity plays a far more important role in this defect than most investigators are willing to admit. I think the reason for the unwillingness to accept heredity as a major factor is the extreme difficulty in obtaining a satisfactory family history of defective vision in one eye, for it may manifest itself in widely varying intensities. Unquestionably, other factors are concerned, such as overaccommodation and resulting convergent strabismus, that leads to monocular suppression and abnormal retinal correspondence.

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Once the diagnosis of strabismus is clear and amblyopia is suspected or, as in older patients, certain, I certainly agree that we should try to do as much as possible with the child and attempt to achieve the primary goal. Many methods are used with varying degrees of success and varying degrees of permanence. The technician and physician must consider carefully the extent of treatment done for each individual.

I think the most important question the technician and physician must answer is "What is going to be the extent of this child's disability as a result of his amblyopia?" If they are not successful in achieving the ideal, how badly will it affect the later life of this child? Secondly, they should ask "If we do not achieve marked improvement in the first three months, both as to visual acuity and stereopsis, are we justified in prolonging the attempt?" I am assuming now that any surgical procedure that is contemplated will be done to realign the eyes. I am thinking only of macular perception. I think that many times far too much emphasis is placed on what the child ought to see, and not enough on what he does see. Amblyopia, or monocular vision, with suppression, inconvenient as it would be for us who normally have stereoscopic vision, is no particular trouble to the individual lacking it from early childhood. He has never known anything else and has learned to adapt himself to his surroundings. Manifest strabismus bothers him because his con-

temporaries tease him, and if he has a high refractive error he may be uncomfortable until it is corrected properly; but if he has one good reading eye, his life need not be jeopardized in any way. In fact, if we dwell too much on his disability, we may produce a frustrated attitude on the part of the patient simply because he just cannot make that bird go in the cage.

After a reasonable attempt with all tricks tried, training, for some patients, should be stopped, and stopped without a fuss. Some occupations, it is true, will be closed to this patient, but he need not live the life of an invalid. This child does not need sympathy or overprotection; he needs guidance and encouragement to accept life as it is and to live it on its highest planes. One of the world's outstanding geneticists has an amblyopic eye. A successful surgeon not only sees well with only one eye, but has had only one eye since before he entered medical school.

The physician and the orthoptic technician can get too engrossed in trying to achieve an ideal that in many instances is neither possible nor of any great moment as far as the future life of the individual is concerned. Poor binocular vision and amblyopia are undesirable but, on the other hand, seldom tragic, and although we like to see good results, I think we can try too hard sometimes. We need not feel that we are doing the patient an injustice when we recognize the impossible.

SURGICAL OVERCORRECTION IN CONVERGENT STRABISMUS

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BEFORE discussing the factors which might result in the surgical overcorrection of convergent strabismus, I will review the types of operative procedures which have been advocated for the treatment of esotropia in general and evaluate the basic advantages and disadvantages of each. From the standpoint of orthoptics, the treatment of alternating esotropia is often more vital than that of predominantly monocular squint, inasmuch as the treatment of the latter is more often aimed at a cosmetic rather than a functional cure. This discussion, therefore, primarily relates to the surgical treatment of esotropia in which approximately equal vision exists in each eye, and to the factors which may cause overcorrection and resultant exotropia.

The first documented operation for strabismus, of any type, was performed by Diffenbach in 1839. He completely severed the tendon of the medial rectus muscle in a young individual with convergent strabismus. Following this procedure, numerous free tenotomies of the medial rectus were accomplished in similar cases, but the operation soon fell into deserved disrepute because of the high incidence of postoperative exotropia and the poor cosmetic result due to posterior displacement of the caruncle.

At the turn of this century it was generally conceded that the surgeon should never operate on the medial rectus muscle, and that the relative position of the eyes should be altered by approaching the lateral recti only. Obviously, this represented an inadequate approach in numerous cases.

In 1922, Jameson⁷ first outlined the principles of his so-called recession operation. Basically, recession is similar to free tenotomy, in that the muscle is retracted and shortened, and its inherent capability for action is therefore decreased. However, in recession, there is a fixed, measurable, and reproducible end point. In 1931, Jameson⁸ elaborated on this technique and, in addition, furnished somewhat more precise data regarding the relationship between the amount of recession and the results which might be expected therefrom.

At present, most ophthalmologists seem to agree that the basic surgical procedure to correct esotropia is medial rectus recession, either by itself or in combination with a simultaneous attack on another muscle. There are a few exceptions to this; Dunnington and Regan,⁴ for example, advocate bilateral lateral rectus resection in those cases where heteronymous fixation exists, and Gibson⁵ and Martin-Doyle¹⁰ favor marginal myotomy of the medial recti in early cases or for patients with a minimal degree of alternating esotropia.

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Generally speaking, however, any debate relative to the type of operation to be employed centers less around whether or not one should recess a single medial rectus than to what degree it should be recessed; and, at the same time, which muscle or muscles should be simultaneously attacked. In a predominantly monocular squint, with greatly lowered visual acuity in the non-fixing eye, medial rectus recession or this procedure combined with a resection of the homolateral lateral rectus is the usual procedure of choice. In the case of alternating esotropia, there seems to be considerable disagreement about the technique which should be employed.

Costenbader and Bair³ have advocated simultaneous binocular surgical treatment of alternating strabismus. They argue that this procedure is more likely to result in postoperative comitance, and further, that comitance is extremely important in the achievement of a good functional result. They also stress the decreased incidence of facial asymmetry following binocular procedures as opposed to surgical treatment of one eye alone. Chandler² states that monocular recession-resection sometimes results in residual esotropia for near combined with exotropia for distance, and he also advocates binocular surgical procedures.

Schlossman and Shier¹¹ have expressed quite well the arguments favoring monocular surgical treatment of alternating esotropia. They acknowledge the fact that a better cosmetic result may be obtained from a binocular procedure, but also emphasize the fact that monocular operations provide greater flexibility. If further surgical procedures are necessary, they can be performed on an untouched eye; in addition, the surgeon may then be guided, to a certain extent, by the postoperative result of the preceding operation.

Determination of the near point of convergence (NPC) as an important factor in the choice of operative procedure has also formed the subject for considerable discussion. Burian¹ feels that it is of little or no importance, because the medial rectus is a relatively strong muscle and, with sufficient stimulus, it should always be able to perform the necessary degree of convergence. The opposite view has been taken by Scobee,¹² who stated that if the NPC is more than 50 mm. from the bridge of the nose, the surgeon should never simultaneously recess both medial recti no matter what degree of deviation is present preoperatively.

At the motility clinic of the University of California Medical Center, the surgeons generally perform symmetrical surgical procedures in any case in which both visual acuity and monocular rotations are about equal between the two eyes. The NPC is determined insofar as is practicable, but it does not play a major role in the choice of surgical procedure. This is largely because of the numerous difficulties and resultant inaccuracies in its measurement, particularly in the case of younger patients. Somewhat arbitrarily, the surgeons have correlated the degree of recession with the degree of preoperative deviation and have divided it equally between the two eyes.

CLINICAL STUDY

In recently reviewing the case histories of those patients who had been examined in the motility clinic, and who had subsequently been surgically treated for alternating esotropia, one rather outstanding fact presented itself. Surgical overcorrection, resulting in exotropia, was extremely prevalent following bilateral 5 mm. medial rectus recessions. During an arbitrary period of time, 1950 to 1954, there occurred, purely by coincidence, 17 each of bi-

lateral 5 mm. and 4 mm. recessions. In the 5 mm. group, 41 per cent of the operations resulted in exotropia; in the 4 mm. group there were no instances of surgical overcorrection. Obviously, there is no long-term clinical follow-up on any of these cases, but there is still a rather sharp line of demarcation between the results obtained after essentially similar procedures which differed minimally as regards their extent. This rather sharp dividing line suggests the existence of a theoretical boundary between a safe and a dangerous limit as regards the degree of surgical correction.

Seventeen patients were treated with bilateral 4 mm. recessions. The age of the patients ranged from 1 to 6 years; the average age was 3 years. The original deviation was 49.4 prism diopters. The change in the relative position of the eyes was 3.8 prism diopters per millimeter of recession. The average post-operative deviation was 20 prism diopters of esotropia, representing a fairly good result. There were no instances of overcorrection.

Seventeen patients were treated with bilateral 5 mm. recessions during the same period. The average patient age was about the same as in the 4 mm. series, roughly 3.8 years; the ages ranged from 1.5 to 8 years. The original deviation averaged 42.6 prism diopters, but the total correction was 47.5 prism diopters. The correction per millimeter of total recession was 4.7 prism diopters. Obviously, overcorrection must have occurred in many instances. The diopters of correction per diopter of original deviation was 1.17 as compared with 0.68 for the other series. Seven cases, or 41 per cent of the total, developed post-operative exotropia.

DISCUSSION

Analysis of both series of cases furnishes a basis for a discussion of the theoretical concepts which are involved

in the surgical treatment of esotropia, and it also suggests certain conclusions. Some of these appear to be in direct conflict with other thoughts which have been expressed on the subject.

On purely theoretical grounds the surgeon can fairly closely predict what should occur following an alteration in the positioning of the extraocular muscles with respect to the center of rotation of the globe. Lancaster⁹ pointed out that in an estimate of the degree of muscle shortening or lengthening which should be accomplished in order to produce a shift in the optical axis of the globe, the surgeon should keep in mind that the operation affects not only the surgically treated muscle but also its direct antagonist, and to an approximately equal degree. This occurs because, in the over-all analysis, the resting tension of any muscle is increased when it is stretched and is decreased to an approximately equal degree when it is shortened. Equilibrium following recession obviously is regained when the force exerted by each muscle becomes equal, that is, when the length of each muscle has decreased by the same proportion. In other words, the net result of a single recession is, theoretically, rotation of the globe to the extent of about one-half the amount of the recession. It can be shown mathematically that 1 mm. on the surface of the globe represents about 4.5 arc degrees (approximately 9 prism diopters). A bilateral 5 mm. recession, for example, represents a change in the relative position of the visual axes by one-half of the total amount of recession. This represents a shift in relative position of 5×9 , or 45 prism diopters.

These theoretical prognostications do not always agree with the actual results following surgical operation on eye muscles. First of all, the relative differences in elasticity between agonist and antagonist have been ignored.

Secondly, anatomical entities such as check ligaments and aberrant muscle attachments influence results. And finally, the secondary action of the various other extraocular muscles, which play an important role in determining the resultant position of the globe, must be considered. All these variables conspire to produce somewhat unpredictable consequences; the correlation between clinical and theoretical outcome is really somewhat surprising.

The viewpoint diametrically opposite the theoretical was held by Scobee,¹² who maintained that the degree of correction obtained by any operation on an eye muscle was independent of the degree of surgical operation and was determined solely by the degree of the original deviation. According to his premise, he performed 6 mm. recessions only. On purely logical grounds there is undoubtedly a happy medium between the two extremes; this has been well expressed by Sugar,¹³ who states that, although the relationship between the degree of surgical operation which is required and the amount of preoperative deviation may show considerable variability between individuals, there would seem to be a general over-all pattern which correlates the two. The theoretical results, compared to Sugar's and those of other observers, indicate that this is the situation.

Table I shows a comparison between the theoretical change of position of the global axes, and the change observed after various surgical procedures. A relatively good correspondence exists between the two.

ETIOLOGY OF SURGICAL OVERCORRECTION

The underlying cause of surgical overcorrection in strabismus may be based upon any one, or a combination, of several distinct factors. Conceivably, the poor result could be based on an inadequate, variable, or careless preoperative evaluation; however, this is probably of minimal importance in any sequential series of cases in a large clinic, because of the comparable and controlled examinations done in each case. By the same token, the factor of surgical technique may also be disregarded because, in the average case, the procedure is standardized, in spite of the fact that different operators may have performed the actual surgical operation. Postoperative changes secondary to surgical trauma, or due to the inexactitudes of surgical procedures in general, are probably of minimal importance also. Gifford⁶ has shown that in most cases a recession of the medial rectus muscle produced a new insertion either at, or very close to, the intended position.

Overcorrection, in those cases where bilateral medial rectus recession has

TABLE I
COMPARISON OF RESULTS IN OPERATIONS FOR ESOTROPIA

1. Theoretical (bilateral 4 mm. recession)	36.0Δ
2. Theoretical (4 mm. resection — 4 mm. recession)	36.0Δ
3. Tour and Asbury (bilateral 4 mm. recession)	30.0Δ
4. Tour and Asbury (bilateral 5 mm. recession)	47.5Δ
5. Dunnington and Wheeler (10 mm. resection — 4 mm. recession)	40.0Δ
6. Cordes and Aiken (O'Connor cinch — 4 mm. recession)	41.0Δ
7. Sugar (4 mm. resection — 4 mm. recession)	40.0Δ
8. Scobee (bilateral 6 mm. recession)	47.0Δ

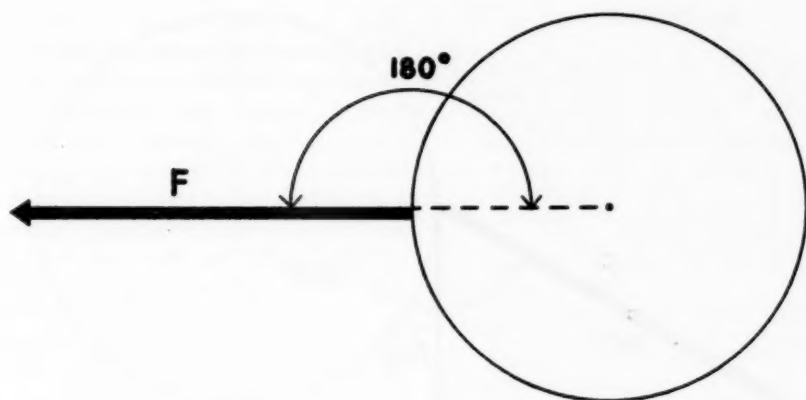


FIG. 1—Force (F) acting along radius of curvature. No torque.

been performed, is probably due to an overenthusiastic surgical approach in which the muscles have been allowed to shorten beyond the point of their maximum effectivity. Because of the

wrap-around relationship of the extraocular muscles and the globe, which results in the placement of muscle insertions somewhat anterior to their point of tangency, relatively small changes in the rotational position of the eye do not alter the direction of action of the muscles but only their effective length. As mentioned before, the results of this shortening can easily be foretold. On the other hand, the point of tangency of all of the rectus muscles lies somewhere near the global equator and, because of this, no muscle should be recessed behind this point. If the muscle were recessed behind this point, its direction of action would be changed and its effective torque would decrease (figs. 1-3). With regard to the medial rectus, however, the point of tangency is about 2 mm. anterior to the equator; it should never be recessed as much as any of the other muscles can be recessed. Recession of more than 5 mm. does not allow the muscle to act at maximum efficiency in adduction, and an increase in inward rotation further rapidly decreases its mechanical effectivity (fig. 4).

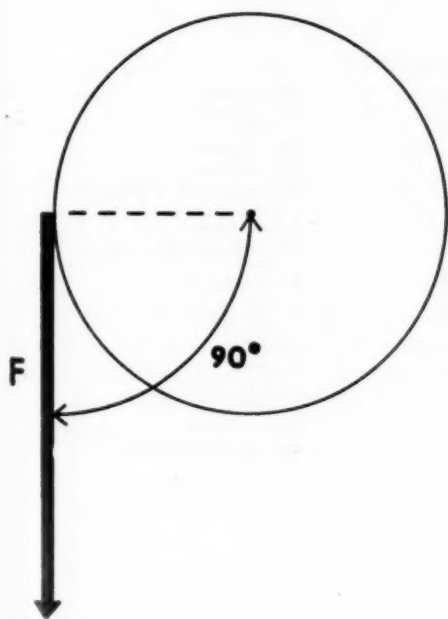


FIG. 2—Force (F) acting tangentially at point of contact and normal to radius. Maximum torque.

In addition, should the medial rectus insertion be located further from the

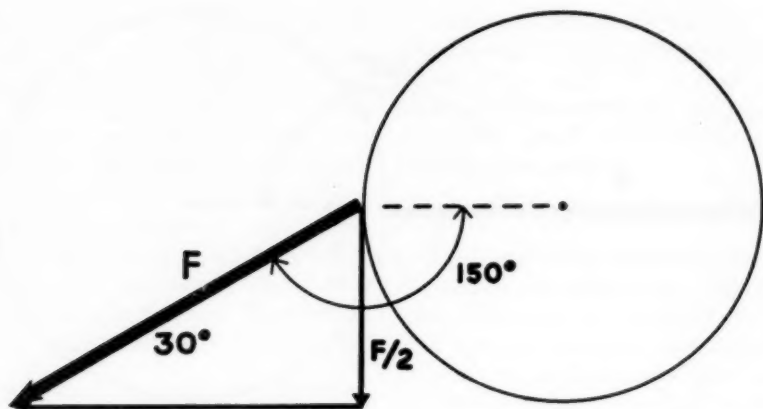
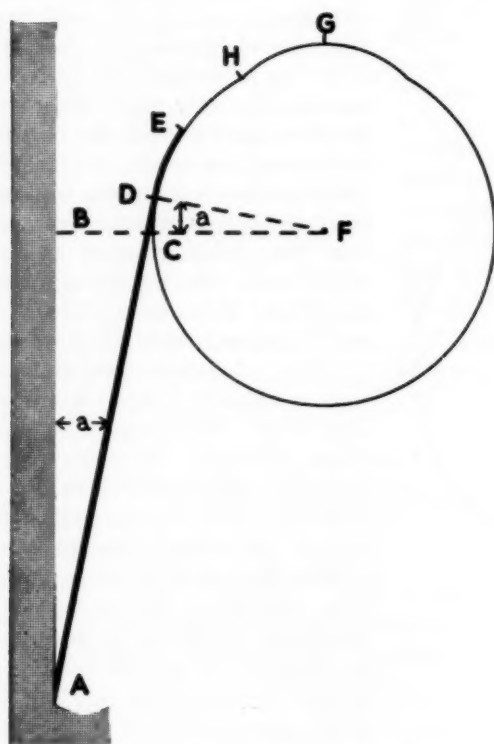


FIG. 3—Force (F) acting at 30 degrees to radius at point of contact. Torque component is $F/2$.



$$\begin{aligned}
 GH &= 6 \text{ mm.} \\
 HE &= 5.5 \text{ mm.} \\
 \therefore GE &= 11.5 \text{ mm.} \\
 GC &= 18.8 \text{ mm.} \\
 EC &= GC - GE = 7.3 \text{ mm.} \\
 AE &= 41 \text{ mm.} \\
 AC &= AE - EC = 33.7 \text{ mm.} \\
 BC &= 6.5 \text{ mm.} \\
 \sin a &= \frac{BC}{AC} = \frac{6.5}{33.7} = .19 \\
 a &= 11^\circ \\
 \sin a &= \frac{CD}{CF} = \frac{CD}{12} = .19 \\
 CD &= 2.3 \text{ mm.} \\
 \therefore DE &= EC - CD = 5 \text{ mm.}
 \end{aligned}$$

FIG. 4—Relationship of medial rectus muscle to globe. Point of tangency (D) is 5 mm. behind normal insertion.

limbus than average, even a 5 mm. recession may prohibit the muscle from acting at maximum efficiency against its direct antagonist, the lateral rectus. Exotropia may logically result in many such cases. On the other hand, should such hyperweakening occur following a monocular procedure, parallelism of the visual axes can still be maintained by increased adduction of the fellow eye, or by turning the head away from the side which has been surgically treated. If the procedure has been performed on both eyes, no such solution is possible.

SUMMARY AND CONCLUSIONS

On the basis of theoretical considerations, as well as clinical observations by both myself and others, it seems logical to draw the following conclusions:

1. Surgical correction of alternating convergent strabismus by means of bilateral medial rectus recession is a fundamentally sound procedure. It results in greater likelihood of postoperative comitance as well as the better possibility of a functional cure.

2. Surgical overcorrection resulting from this procedure should not be considered as a fault of the operation itself, but more likely occurs following an overenthusiastic approach to the situation on the part of the operator.

3. The safe limit of a single medial rectus recession is probably in the neighborhood of 4 mm. The degree can be determined on theoretical grounds alone, and seems to agree quite closely with the clinical findings of numerous investigators.

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CONSIDERATION OF THE WHOLE CHILD WITH STRABISMUS

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THE consideration of the whole child in the treatment of strabismus is placing emphasis upon the obvious; such emphasis is helpful at times.

It may be amiss to speak of the whole child to trained orthoptists. Orthoptists have led the way in this consideration and have reminded the doctor of such facts concerning his patients with strabismus.

The literature contains many references to nonocular factors related to the etiology, diagnosis and treatment of strabismus. Scarcely anyone writing in this field has failed to devote paragraphs or chapters to this subject, but the detailed descriptions usually take these related elements for granted. The effort of the ophthalmologist, the orthoptist, the family, and even the patient may be jeopardized or nullified by inattention to the environment of the individual patient.

Among other medical means leading the way in this concept is the development of training centers for mentally handicapped children. Dr. Esther B. Clark of Palo Alto, in speaking of the Children's Health Council of that city, states, "We must treat the whole man, body, mind, and spirit." She adds, "An integrated program of total rehabilita-

tion, emotional and physical, is necessary."

The "whole child" is a complex concept. It includes his parents, his teachers, his siblings, and the children with whom he plays and fights. It may include his grandparents. To be considered are the factors concerning the child's home, and its hygienic practices. The concept includes the child's heredity, both physical and mental. It includes his general health, growth, and progress. Our consideration must encompass everything concerning the child's nature and environment, as well as the specialistic examination of the ocular status, if we are to treat strabismus. It is another way of saying that we have no definite etiology of squint, and recognize that all such factors may play a role in the diagnosis and treatment of this disorder.

Kramer⁶ states that an individual's ability to develop binocular skills depends on several factors, and includes among these the following:

1. *Heredity* including anomalies, nervous instability, physical or mental retardation, and underdevelopment.

2. *Physical Development.* The presence or absence of general good health and stamina must be emphasized in orthoptic training. The child must have plenty of outdoor exercise, proper rest, nourishing food, and practice good habits of hygiene. A child that is under-

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weight and has anorexia and low resistance to disease is not a good subject for therapy for strabismus.

3. *Mental Development.* A child should have a mental stature capable of moderate spans of attention and concentration if specialized training is to be of any value.

4. *Psychological Development.* Each patient with strabismus presents a psychological problem which must be studied and handled with tact. The physician should know the family background and have some knowledge of whether the child is a member of a happy or an unhappy family group. The child's adjustment to his playmates in his home and school environments should be considered. Miss Kramer adds that the unknown nervous factors may be the direct cause of intractability in children and that as soon as some of these factors are improved by treatment a new personality may be produced for the child.

Forbes⁴ has stated that comparatively little thought has been given to the mental effect which a deforming squint has on a child, and this effect may far outweigh the ocular problem. A child becomes conscious of his difference from other children and, subsequently, often develops inferiority impressions, mental depression, and signs of withdrawal or aggression. Such reactions must be actively opposed. The child, his parents, and his teachers must realize that he is like other children.

Approaching this subject from another viewpoint, Anderson¹ lists problems in the treatment of strabismus and includes in his listing the following: (1) lack of parental interest; (2) difficulties arising if treatment becomes a greater handicap than the ocular deviation; and (3) the difficulty and effort required in knowing what a child sees and means.

There is no way to be dogmatic concerning these problems, and most of us approach them according to our own personality and training. In the first place, the physician must have a regard for children if he is going to treat the whole child. Those ophthalmologists who limit their practice to children set an excellent example for the general ophthalmologist to follow. The associated orthoptic facilities enjoyed by many ophthalmologists are advantageous. This contribution of orthoptics is not publicized as much as it should be. Attention to these many factors is a time-consuming process requiring extreme patience. Any description of the methods is open to controversy.

History-taking is an important non-ocular factor. Try to get more exact knowledge about the onset. Was there any illness or injury at the time of onset of the squint? Were there any frightening experiences or shock occurring before or at this time? The physician should explore the question of behavior changes, the relationship with siblings and playmates, and the home situation, including the question of changes of homes in relation to onset.

The history of systemic disease and any indicated examinations and referrals are important. If systemic history indicates any likelihood of disease or congenital defects, the child is referred to the pediatrician for examination. Children are examined for any nose, throat, or ear complications, and blood counts taken to rule out infection or anemia, if such seems indicated. Ophthalmologists are especially interested in the evidence of any generalized or hereditary diseases, any related developmental anomalies, any functional defects, and any psychological factors indicating maladjustment to the environment. If mental retardation is suspected, psychometric evaluation may be requested.

On the first visit the child may be observed while the doctor is talking to the parents and, if old enough, he may be asked to contribute to the history. The main purpose of the examination is to become acquainted with the patient and put him at ease. Besides the ocular and motility examination, an attempt is made to assay the child as to his general health, mentality, hearing, speech ability, and level of concentration. A real attempt must be made at the time of this important first impression to gain the confidence and interest of the child, and not to frighten him in any manner. In the young or very shy patient the examination may afford only meager information, but to enlarge on such information at the expense of future rapport is a mistake. It is important to know whether or not a patient will be difficult to handle. If enough facts are elicited at this examination, a discussion with the parent or parents is in order. Although they understand that the child has had only a preliminary examination, some of the knowledge gained and indications for the future can be broached at this time, remembering not to disturb their confidence in others whom they may have consulted concerning the complaint. The ophthalmologist should also outline the facts regarding the effort, time, and expense involved in the treatment of strabismus. Routinely, the next visit consists of the examination under a cycloplegic, and the parents are given instructions for its use.

Subsequent visits, as necessary, are handled with the idea of stimulating interest in the child. If the father has not visited, he is encouraged to do so in order that he may understand the aims of the therapy and lend his aid. Although it may create difficulties for the busy clinic nurse, the parents are asked to bring any siblings at subsequent visits, in order that they may be surveyed for

related eye signs. In the average case no effort is made to exclude the parents from the room. Young patients are more at ease with a parent present, and the parent is more likely to cooperate if she feels an integral part of the examination and progress. However, it is often beneficial to examine the child in the absence of other people and this can be interpreted as a recognition of his progress.

If facts of the case are discussed with the parent, no attempt to exclude the child is made, excepting in the rare instance when he is extremely excitable or when repeat surgical procedures may be first mentioned. The presence of a sibling may stimulate the child to try even harder, and at times a strange child brought into the room will quiet a frightened or unruly patient. If the child is admonished, it is better that the doctor do so and that the parent refrain from such corrections, unless there is naughtiness which threatens family patterns. Usually patients are tractable when asked to be so, and extend their periods of attention and cooperation if allowed occasional freedom and relaxation. Anderson¹ has stated this more succinctly by denying to the ophthalmologists and orthoptists the role of a disciplinarian. He urges that the doctors and technicians try to meet the moods of the child, and warns that personality cannot be altered in the brief periods of treatment.

The ophthalmologist may dispel some of the patient's fear of hospitals and doctors, if he has such fears. Doctors should not remove hospital uniforms, although it is stated that such clothing frightens many children. It seems better to prove to the child that people in hospital uniforms need not cause hurt. Much has been written about rewards, and it is true that this practice may calm a patient. The child that is slightly more mature is better handled by

praise or some visible indication of progress. Results are proportional to the interest of the physician in the patient, as well as to his technical maneuvers. Answers to the patient's questions need to be more erudite than he might judge by reference to the questioner's age. The patient's strabismus must never be spoken of as an abnormal condition.

Any treatment adjuncts carried out at home require careful and repeated explanation to the parents. The need and purpose of glasses are carefully explained. The parents should not discover for themselves that their child with accommodative esotropia sees as well without his glasses as with them. When full corrections are prescribed for such cases, the initial contact with the glasses often determines the readiness of acceptance and must be carried out with care. Cycloplegia should be re-established before such corrections are fitted. The first application of spectacles in fitting the patient with accommodated hyperopia may cause sufficient blurring to initiate a dislike of the correction which is difficult to overcome.

Occlusion is so important and so easily mismanaged that it deserves special attention. Janus⁵ warns against occlusion in the nervous child who becomes temperamental and unmanageable. Such an occurrence should be watched for and is contraindication for this treatment. It seems remarkable that children accept total occlusion as well as they do. Little difficulty is encountered as a rule. The very young child may remove his patch at first, but this is usually overcome after two or three days of perseverance. Benzoin tincture may help keep the patch in place in warmer climates. In the "Wheaties set" the use of the patch-club as an incentive is of value. Other children respond more to some tangible indication of their progress. A photograph of the Snellen chart with marks to indicate the progress in

visual acuity has been described by Fowler. In the older child, especially in the teens, the use of total occlusion must be in agreement with the patient's wish and stem from his sincere desire for improvement. Burian² speaks of the importance of motivation in the treatment of amblyopia and cites the remarkable success achieved during the last war, when in order to qualify in certain branches of the fighting forces, young men were eager to improve their vision. The parents and teachers must be urged not to spare the child in his visual tasks through misguided sympathy. The child should be warned against playing in the street and riding wheeled vehicles.

At the beginning of orthoptic training the referring doctor should acquaint the orthoptist with the general factors concerning the case as well as the ocular findings. He should also inform the parents of the aims and nature of this recommended therapy, thus serving as a liaison between the two. At this time any general debilitating factors or diseases should have been improved or corrected. Smith and Kuhn,⁸ although speaking of adults, found that individuals with signs of anemia or any toxic focus of infection, did not respond well to orthoptic training. This graduation to the orthoptic adjunct increases the confidence of the patient and parents in their treatment and ability to progress. In speaking of the selection of patients for orthoptic treatment, Kramer⁶ puts the status of the cooperation of patients and parents in an important place in the factors for such selection.

The orthoptist's approach to the whole child is an extension of the attitudes of the ophthalmologist. Orthoptic methods must demonstrate progress, and only by progress can the patient's interest and cooperation be maintained. No delay in changing the mode of attack should ensue when orthoptic prog-

ress halts, not even to please the ophthalmologist or parents. One attack of apathy and hopelessness in even the young patient is a disease which readily recurs and becomes chronic.

Orthoptic technicians must reduce the age limit at which they can supply some measure of success. Earlier literature contains statements of age limits of 5 years. Now 3-year-olds are treated successfully. Technicians will refine their methods and understanding, gradually lowering the age at which patients can expect physicians to give some diagnostic and therapeutic aid. Middleton and Malcolm⁷ stated:

More is required of a technician than a solid knowledge of physiology and anatomy and the ability to make a correct diagnosis and conduct orthoptic treatment. She must realize that a patient is not just a pair of eyes. He has fears, apprehensions, and other emotions which have to be considered and evaluated along with his strabismus. Most important of all, an orthoptist has to have a real love for people and a desire to understand them; else, how can she happily apply herself to her chosen profession.

When surgical correction is indicated, it is the habit to discuss it with both the parents and the patient. No attempt is made to hide this discussion from the patient, although often the parents are apprehensive about such a revelation. Unless circumstances are unusual, the patient's reactions are more favorable than when the surgical procedure is explained later in the absence of the doctor. Preparation of the child for the operation and attendant procedures is very important in respect to care in the postoperative period and maintaining the patient's cooperation. The confidence of children after surgical treatment is rarely lost if attempts are made to treat them fairly, to mislead them in no way, and to spare them any undue discomfort.

The patient is admitted on the morning prior to the operation. The neces-

sary clearances and physical examination by the pediatrician, together with laboratory work and chest roentgenogram, are completed before entry to the ward. The average child enjoys the new faces and pleasant surroundings of the ward and becomes acclimated to his surroundings by this earlier admission. Many doctors prefer to admit children just prior to surgical operation and discharge them as soon after as they are reactive. In this way the parents can remain with the child. This attitude has many advantages. With longer hospitalization, it is rarely possible for the mother to stay with the child. It is, however, desirable that the parent be allowed some access to the child after he leaves the recovery area and is resuming awareness of his surroundings.

The anesthetist sees the patient the evening before the surgical operation. He prescribes the preoperative medication and, if appropriate, discusses the anesthetic procedures with the patient. Burstein⁸ speaks of the importance of this explanation by the anesthetist. He states that it is necessary to win the trust and confidence of the child, and advises that the anesthetist take the child to the operating room and let him apply the mask at his own volition. Preoperative medication is left to the judgment of the anesthetist. Analgesia before anesthesia is desirable in older children. The method of induction is extremely important. The operation itself should be as nontraumatic as possible, and sutures of absorbable material should be used in the conjunctival closures. After a symmetrical operation no bandages are applied. After cosmetic surgical procedures for older children, the amblyopic eye, if surgically treated, may be patched. Systemic antibiotics are not used unless needed. If they are used, parenteral medication is avoided after recovery from the anesthesia.

The child remains in the hospital

two or three days after surgical operation. This furnishes a time for him to recover from his postoperative discomfort and to have some supervised treatment and observation, and for the surgeon to gain some indication of the results of the surgical correction. If necessary, the child is restrained by elbowcuffs, but otherwise he is allowed freedom of the crib or the ward as time passes. Antibiotic ointment is instilled three times daily, and warm compresses may be started on the second or third day if tolerated. No attempt is made to irrigate the eyes of young children, since this usually is a frightening procedure. Instead, sterile lint moistened with warm saline solution is used to bathe the lids gently. In the absence of bandages there is usually little secretion and stickiness to combat, the eyes are opened much earlier for observation, the child seems to have less disability, and early resumption of orthoptic treatment seems easier.

In summary, emphasis has been placed on the importance of considering factors other than the ocular status in the treatment of strabismus. A possible approach to this consideration of the whole child has been outlined from the

standpoint of the initial history taking and the first examination, the discussions with the parents, the handling of subsequent visits, the management of procedures and treatment at home, the orthoptic phases of therapy, and the surgical procedures, including preoperative care, anesthesia, and aftercare.

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THE FORESEEABLE PROBLEMS IN THE TREATMENT OF ACCOMMODATIVE ESOTROPIA

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THE treatment of accommodative esotropia has not changed significantly in the past ten years. However, the approach to the basic problems has been greatly clarified by clinical experience.

The treatment of accommodative esotropia may vary with the goal to be attained. If the objective is normal fusion with lens correction, only a few problems may arise. If the treatment is carried further, to include dissociation training, the problems increase correspondingly. Along with the various problems are technical, professional and emotional limitations, which should be recognized and considered when evaluating the possibility of a cure.

Essentially, the problems fall into groups, namely, the professional doctor-patient-technician relationship, technical skills and shortcomings, and perhaps most important of all, judgment or the ability to know when and how much to do.

Everything has been said many times regarding the ethical and professional status of the orthoptist in her relations to the patient and doctor. The doctor's attitude, experience and preconceived ideas of orthoptics should be ascertained. The orthoptist will show great acumen in verifying what the doctor has told the patient regarding the aims and purpose of orthoptic training and what it will accomplish in his case. If

the doctor has the positive approach with the technician in what she is to accomplish for the patient, and then takes a negative approach with the patient, with statements such as "orthoptics may help" or "you might try it and see what it can do" or "you will wear glasses for the rest of your life whether or not you have orthoptic training," it is quite conceivable that the patient will not complete the course of treatment necessary for the desired end result.

In any discussion of the treatment of accommodative esotropia, the question of removing glasses always animates conversation. One or more in a group of doctors will ask, "What effect will this (i.e., the enlargement of the relative accommodative amplitude) have on the child when he reaches high school?" "College?" "When he becomes presbyopic?" "Will going without glasses precipitate glaucoma?" An orthoptist serving a group of doctors should ascertain, before treatment is started, how much amplitude of relative accommodation a doctor wishes to have developed in his patient.

Perhaps the greatest single problem confronting the orthoptist is the technical one, utilization of her tools. The master craftsman is one who not only uses the proper tools, but who, through experience, knows how to use them effectively and surely. His knowledge and ingenuity enable him to do the many little things with his tools that

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make his work superior. The finest surgical instruments do not make a good surgeon, a set of fine artist's tools does not create the masterpiece, but without the tools, the surgeon or artist would be greatly handicapped. The objective is a common one, but the means to gain the objective vary according to individual handicaps, talents, skills, knowledge and experience.

Ordinary orthoptic problems need only ordinary orthoptic skill to solve. This is done generally by ordinary use of instruments. But how many orthoptic problems are ordinary? Many different answers could be given to this question. Thinking in terms of the patient, no eye problems are ordinary to him. During what stages of treatment does he experience his crises? Can these crises be prognosticated? Of course they can. The crises have been precipitated by the patient's inability to proceed further along the psychologic, physiologic or economic front. The orthoptist has daily encounters with these orthoptic crises of the patient. A previous study of the patient's potential physiologic, psychologic and visual limitations and capabilities enables the orthoptist to know when measures are to be taken to prepare for or prevent a crisis. These crises occur with total and partial occlusion and in almost any stage of dissociation training.

Little emphasis has been placed on the potential capacity for cure and the human limitations inherent therein. The failure to employ and achieve the full potential working capacity, especially in home training, is often the thin thread between success and failure. How often has the initial enthusiasm and cooperation of the patient and parent been found to be almost limitless, only to vanish as suddenly because of an unforeseen financial crisis in the family or the mother's pregnancy, or the

child's sudden aversion to anything that appears to be routine or work. Motivation plays a large role in child-parent cooperation, but it is not the entire answer. An evaluation of the personality, mental and physical health, intelligence, preconceived notions and prejudices, ambitions and aggressiveness of the patient and parent is often very revealing. The potentialities and patterns for success or failure are inherent in children and adults alike.

Without benefit of complicated and expensive psychologic tests, the orthoptist can recognize these success patterns in the child and parent. The first clue in the chain is the outward appearance. The mother's sense of responsibility or her shiftlessness will be apparent in the child's dress and cleanliness. The second clue is one of attitude. Confidence, optimism and a sense of responsibility are reflected in the mother who always manages to do the things that must be done no matter how many children she has to care for, how no-good her husband is, or the part she plays in the support of the family. An attitude of self-pity, doubt, pessimism and complaint is observed in the mother who is the "perfect alibi" for her shiftlessness or poor management. The child's attitude is the reflected parental attitude. The third clue is that of intelligence and motivation. Vanity is found to be the motivation in most instances, but according to intelligence, it is modified or enhanced by other factors such as ambition, career preparation, fear or aggressiveness. The fourth clue is the emotional stability of the mother and child. It is not too difficult to understand that the more stable the person, the less upset he will experience in the course of treatment.

Since the greatest percentage of patients treated for accommodative esotropia are children, we must consider the parent, usually the mother. Her

intelligence, attitudes, prejudices and emotional stability are as important to success as those of the child. It is stressed before treatment is begun that the mother and child must make the course of treatment a joint career, especially when more than a few diopters of dissociation are contemplated.

The child with allergies, the asthmatic, the child susceptible to hay fever, frequent colds or infections presents the problem of frequent interruptions in the treatment. These children require longer periods of time for dissociation training. Judgment must be used in recommending reduction of glasses, even though the indications are apparent. These children come to the office for training and inspection when they are well and do not necessarily maintain the same amplitude of relative accommodation during their sick periods.

From the foregoing, it is apparent that before the orthoptist advances the fusion therapy in an individual case she must consider all of the facets rather than a few technical ones, important as they may be. Just as one would read the traffic signs and instructions along the roadside when driving an automobile, so should the orthoptist read the problem signs before the problems arise.

Table I is a tabulation of 100 cases of esotropia treated under the direction of Dr. Wade Hampton Miller of Kansas City, Missouri. It is included here with Dr. Miller's permission. It reveals some interesting findings and important clues in orthoptic therapy. The following conclusions were made during the treatment of these patients and from the study of the results.

Control of Suppression By Elimination of Accommodative Stress and by Occlusion

Accommodative stress is eliminated by refractive procedures performed by

the doctor in conjunction with occlusion therapy. This is especially beneficial for patients under age 10 in the over-all treatment of esotropia regardless of origin. In the so-called easy cases, with no appreciable refractive error and with established fusion and no deficiency of relative accommodation, the response to surgical treatment, orthoptic training or both, did not require refractive support to maintain an orthophoric position or to assist in enlarging the amplitude of relative accommodation. With few exceptions, all of these patients were either aged 10 years and older or had attained their peak accommodative power. However, they did need anti-suppression aids in the form of partial occlusion and orthoptics.

The problems in the treatment of suppression are manifold. First is the problem of eliminating amblyopia. Relatively few patients with amblyopia remain unimproved, even after age 7, if adequate amblyopia therapy is carried out at home.

Human nature being what it is, more frequently no mention will be made of the inefficiency with which the active home training is carried out or the poor cooperation on total occlusion. The best defense is a good offense, and the orthoptist should not hesitate to point out the reason for lack of progress when the patient comes in for inspection.

These patients need close supervision and should be inspected for progress or regression every two weeks. Encouragement, pep talks, and sympathy should be included in the instructions given to the patient and parent. Only close supervision can avert an upset or crisis during the amblyopia therapy and make the improved visual result more gratifying. However, if a crisis cannot be averted even with close supervision, and the pressure to remove the total occlusion has reached a critical point,

it seems good judgment to supplant the total occlusion with partial occlusion, after explaining to the patient and parent that this can be done but that it will make progress considerably slower and the orthoptic training period longer. The decision should be theirs to make; a harmonious home life may be more important to that family than a quick cure for the child. Whenever possible, total occlusion is not interrupted until the amblyopia is reduced within one line difference between the two eyes with full hyperopic correction; better yet, if the difference is only a few letters.

Partial occlusion often presents greater problems than total occlusion, especially if the suppression is marked and the hyperopia not fully corrected for orthophoria-emmetropia. The patient will often ask that total occlusion be resumed. In these instances, total occlusion is resumed until the suppression has decreased to a more tolerable binocular situation.

The type of partial occlusion is of utmost importance to the well-being of the patient. When total occlusion is replaced by partial occlusion, it is well to use a translucent tape over the lens, or the commercial blur lens, through which only light is transmitted. Lesser degrees of partial occlusion with clear nail lacquer are gradually introduced according to the amount of suppression and residual amblyopia present.

A good rule to follow in determining when to lessen the blur is to wait until macular suppression has been overcome before removing the tape occlusion for a more transparent type. The standard gross tests, such as the Worth four dot test, are inadequate for screening purposes. The major amblyoscope with suitable slide material, i.e., small test objects with foveal and macular controls not common to both eyes, seems to provide the most adequate screen test for the control of suppression.

After macular suppression is brought under control, nail lacquer sufficient to blur the vision of the fixating eye one line less than the suppressing eye is used. The lacquer must be applied with care, since waviness in the coating creates distortion, which in turn adds confusion to the existing state of binocular confusion for the patient.

Most patients complain bitterly of partial occlusion. This complaint is a justifiable one, especially if the suppression is marked and the glasses do not maintain orthophoria for the patient. Here is a problem that should be anticipated so that it can be explained to the patient and he is in some measure prepared. As the suppression gradually lessens, the complaints decrease. I find that the greater the suppression the greater is the complaint against partial occlusion. Generally, it is only when the macular suppression is finally eradicated that the patient tolerates partial occlusion to the point of indifference toward it. This is a sure sign of progress, assuming that the partial occlusion has been properly applied. This unhappy period is always anticipated, just as lacrimation and irritation result in the first efforts to fuse through voluntary accommodative control.

Foveal Suppression

Control of foveal suppression means success in the treatment of accommodative esotropia, since it occurs with accommodative stress. The greater the accommodative stress, the greater is the foveal suppression. Therefore, foveal suppression is controlled in a dual manner through refractive measures and partial occlusion. At this stage of treatment the doctor and technician must work jointly and in close harmony.

1. The doctor renders the patient emmetropic with adequate correction of the refractive error. Adequate

TABLE I
TABULATION OF FINDINGS IN 100 CASES OF ESOTROPIA

ETIOLOGY	Accommodation, 90-100% with accompanying vertical deviation of the dissociated type paretic or others	12 6 6	57
	Mechanical with accompanying vertical deviation	0	3
	Mixed accommodative-mechanical-paretic with accompanying vertical deviation of the dissociated type paretic or others	13 3 10	35
	Paretic with accompanying vertical deviation of the dissociated type paretic or others	2 0 2	5
	Congenital with other stigmata with general spasticity with nystagmus	2 0 2 of the accom. group	2
*FUSION STATUS	No fusion		9
	Grade III suppression (foveal-macular-peripheral)	includes est. ARC's	34
	Grade II suppression (foveal-macular)		40
	Grade I suppression (foveal)		17
DOMINANCE	True alternators (all in the no fusion status)		5
	Alternators with preferred dominance		10
	One-eyed dominance, not marked		40
	One-eyed dominance, marked		45
AGE IN YEARS	7 and under		48
	Between 7 and 25		35
	Over 25		11
	Over 40 or presbyopic		6
AMBLYOPIA of 2 or more lines difference	Under age 7		19
	Over age 7		26
REFRACTIVE STATUS	Hypermetropia		93
	Myopia		7
**COOPERATION	Completed cases		67
	Under treatment (supportive antisuppression, refractive)		12
	Poor cooperation (9 parents, 2 patients)		11
	Dismissed as incomplete (classified as 80% cured)		10

*The fusion status is graded according to the suppression since the treatment is directed to the suppression.

**Eighteen patients seen during this time interval did not remain to begin, continue or complete treatment. They are not included in the above tabulation.

TABULATION OF FINDINGS IN 100 CASES OF ESOTROPIA

TREATMENT			
	Surgical only (all under age 2¾)		2
	Orthoptics only (all over age 7)		5
	Refractive, antismpression only (all under age 3)		3
	Orthoptics and refractive without add for near under age 7 over age 7 with add for near under age 7 over age 7	3 13 38 27 11	54
	Surgical treatment and orthoptics with refractive support without add for near under age 7 over age 7 with add for near under age 7 over age 7	0 5 18 13 5	23
CURE	Complete cure (with normal binocular vision) without the assistance of glasses (hyperopes) or with glasses (myopes) under age 7 over age 7	24 15	39
	90% cure (continuing on supportive measures of partial correction of hyperopia with partial occlusion of the dominant eye to control suppres- sion) under age 7 over age 7	7 11	18
	80% cure (constant wearing of glasses with partial occlusion to maintain control of amblyopia and suppression with tendency to Et)		10
	Fair functional improvement 20-80% (poor cooperation, too much ambly- opia, surgical treatment indicated but not accepted were the prominent causes of failure)		11

AVERAGE LENGTH OF TIME UNDER TREATMENT

FUSION:	Months				Years			Months				Years		
	3	6	9	12	1½	2	3	3	6	9	12	1½	2	3
Grade I supp. (17)	2	5	0	0	0	0	0	8	2	0	0	0	0	0
Grade II supp. (40)	4	8	6	1	0	1	0	8	6	2	2	0	2	0
Grade III supp. (34)	0	6	8	0	0	1	0	0	6	5	3	0	3	2
No fusion (9)	0	0	0	0	2	3	1	0	0	0	0	0	2	1
	Age 7 and under							Over age 7						

correction is the lens power necessary to relax the accommodation to almost complete passivity for both distance and near. Of the cases listed in table I, 38 out of 54 patients had bifocals of +3.00 spheres cemented on for relaxation of the accommodative effort at the near working range. Sixteen did not require the additional correction for near; 3 of this group were under age 7, the remainder over age 7, or at their peak accommodative power, which, according to Donders, is around age 10.

2. The patient is made orthophoric, if possible, irrespective of the fusion status. Orthophoria was achieved for 54 of the 77 patients studied by complete relaxation of the accommodative effort with the maximum hyperopic correction, both for distance and near; also the adequate correction of any existing hyperphoria. Here again, those who could not maintain the orthophoric position for near with accommodative stimulus were given the necessary additional correction for near. Twenty-three of the 77 patients needed the next step, an operation, to obtain orthophoria.

3. Surgical correction is done to render the emmetropic (with glasses) patient orthophoric, when complete relaxation of the accommodation by refractive measures did not accomplish this. In the series studied, surgical correction was not done until the patient had worn the full correction for a minimum time of one month. A slight surgical overcorrection was found desirable for the orthoptic regime to follow. When the esotropia was accompanied by a vertical rectus paresis or weakness, surgical overcorrection of the esotropia occurred.

The effect of the operation in the 9 cases with no fusion was good when complete relaxation of the accommodative effort by refractive measures was maintained. In 5 of these cases, orthophoria

was established for both distance and near with one surgical procedure and with the additional refractive support to maintain orthophoria. With the eyes in this position, all of these patients developed fusion, though at a much slower tempo.

4. Reduction of the lens power quickly followed the operation. This proceeded at a more rapid pace with the surgical cases than it did with the non-surgical cases. The power for near was reduced first, 1 diopter at a time. In some cases the entire additional near power would be reduced before the distant correction was reduced. In others, the near power would be reduced to 1 diopter, then a reduction in over-all or distant correction before another or final reduction in the bifocal power. Patients who had fusion responded, whereas the cases with no fusion could not tolerate reductions without a breakdown until after peripheral fusion was established. Generally, it was more desirable to allow the correction to remain longer than necessary than to err by reducing it prematurely; correction should remain until the relative amplitude of accommodation is sufficient to undertake added responsibilities.

Therefore, one of the big foreseeable problems can be eliminated by adequate control of the accommodative stress through adequate refractive procedures, especially in patients under age 10, regardless of cause. The orthoptist can be of real assistance to the doctor in making the necessary evaluations on the fusion status during this period.

The patients who benefit by control of the accommodative stress through refractive measures fall into two main groups.

1. Under age 7. The greater the suppression, the greater need for refractive measures to maintain the ortho-

phoric position of the eyes. The need for supportive refractive measures was less urgent in the age group between 7 and 10. After that age, or when the accommodative peak had been reached, it was uncommon to find this procedure necessary except to follow normal refractive measures. Complete relaxation of the accommodation was not necessary. These patients were all able to demonstrate diplopia and had established fusion prior to treatment.

2. The cases of no fusion, in which, without refractive support, neither orthoptic training nor surgical correction satisfactorily benefits the patient. Only small refractive errors were found in these cases. Of the 9 patients without fusion, all were alternating in the beginning, but as they proceeded to a fusion status, a preferred dominance of one eye became increasingly apparent. Complete relaxation of the accommodation both for distance and with +3.00 spheres added for near was necessary, and had to be continued for a much longer period of time than in those patients who had established fusion.

CURE

The adult was cured in less time than the child under age 10.

In the cases with pre-established fusion, the patient would arrive at a functional cure in three to six months, depending on the amount of hyperopia and control of the foveal suppression. All supportive refractive and antisuppression aids could be eliminated in approximately one year. With few exceptions, the greater the amount of hyperopia, anisometropia, amblyopia-suppression, the longer it would take to establish a functional cure. The patients with no fusion required from one and a half to three years. They were unable to develop foveal fusion. Only 4 patients could demonstrate

macular fusion under controlled conditions of the major amblyoscope. The entire group obtained peripheral fusion.

In the cases where a surgical procedure was part of the corrective treatment, the refractive support could be eliminated and a functional cure established at a much faster rate than in those treated with nonsurgical measures alone.

CONCLUSIONS

Control of suppression during the training of binocular skills is the greatest single problem that confronts the orthoptist. Suppression should be checked at all sessions to note whether the refractive support is sufficient to maintain orthophoria and, also, whether the partial occlusion is adequate to maintain binocularity or fusion.

In accommodative esotropia, foveal fusion occurs with accommodative stress. Therefore it is not an isolated problem. When the accommodative stress is removed through proper refractive support, the foveal suppression is eliminated or greatly lessened. Partial occlusion should always accompany the refractive support. Thus, the presence of foveal fusion is an important clue to check the adequacy of treatment, whether refractive or orthoptic.

Knowing when to reduce or add to the over-all dioptric power of the patient's corrective lenses shortens the length of treatment and oftentimes avoids a crisis for the patient. Proper timing of these procedures to the relative accommodative amplitude often achieves dramatic results.

The correlation of refractive procedures to the amount of suppression and relative accommodation enhances the effectiveness of simple orthoptic methods by:

1. Reducing the tropia wholly or partially, depending on the status of the relative accommodation

2. Proceeding from a surgical result to a functional as well as a cosmetic cure

3. Maintaining and strengthening the habit pattern of binocular stability during the varying stages of fusion training

4. Maintaining straight eyes in the classical no fusion cases, with a striking development of fusion

The study of the foregoing cases would indicate that possibly the learning aspects of orthoptic training can be overemphasized. Single binocular vision occurs as a natural development in the child under age 7, providing the climate for orthophoria or near orthophoria is

established by surgical correction or proper control of the accommodation by refractive procedures, or both, along with careful supervision and control of the suppression. Orthoptic training can and does enhance the binocular skill the child would develop naturally under favorable conditions.

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THE FUNCTIONAL FACTOR IN CONVERGENT STRABISMUS

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FUNCTIONAL factors affect strabismus in many ways and I shall discuss a few of the more common groups. First, the effect of functional factors on causation; second, the effect on recurrence; then the effect on motivation leading to a physiologic cure; and, lastly, the effect of the functional factors caused by the strabismus—in other words, the effect of the strabismus on the individual.

One of the most enthusiastic authors to write on the psychologic aspect in relation to strabismus was W. S. Inman.⁴ He became interested in strabismus because of his interest in child psychology and thought that the deformity was in the nature of a hysteria. Since he was unable to obtain evidence of mental stress from the children (age 5 and younger), he began to study the families concerned. A consecutive series of 150 family case histories was investigated, and he found a definite relationship between squint and lefthandedness and stammering. Relative to the psychologic aspect, he found as an outstanding feature of these cases a frequent history of parental strictness of an oppressive nature.

There have not been very many authors who have so wholeheartedly supported the theory of psychogenic causes, and we have some well-re-

spected ones who all but deny such an origin. Burian^{1,2} states that so-called psychological squints, which appear in some classifications of strabismus, belong in the group of purposive strabismus (Chavasse). In these patients the appearance of a manifest strabismus is supposed to be precipitated by abnormal psychological reactions of a child to his environment or by the well known tendency of children to imitate others. Needless to say, these psychological factors can only produce a manifest strabismus where a latent strabismus is already present. And again, "Psychogenic causes for the occurrence of a strabismus are sometimes alleged."

The large area between these two opinions is covered by the rest of us who believe that while emotional upsets may cause a strabismus, such cases are not too frequent. It is this infrequency that makes a review from time to time worth while.

Pugh⁵ has assigned a psychologic cause to 20 per cent of 1,000 cases. In this group she includes jealousy, imitation, fear and shock as the underlying causes. She also states that assigning a definite cause to every case is not easy because of overlapping areas of causation. The patient may be emmetropic or not, and of any age. Occurrence of squint after 8 years suggests a psychologic basis.

Scobee⁶ has pointed out:

The functional factor is a result and at the same time a cause of nervous tension, thus

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setting up a vicious circle. A patient whose accommodative factor has been eliminated by glasses and whose deviation still varies tremendously, usually has a functional factor.

The isolation of a single factor in the vast group of factors that lead to and sustain a strabismus is admittedly risky. However, it is profitable now and then to take a longer look at some items that receive scant attention. In the literature on motility, almost no articles can be found on the functional factors alone. The problem is mentioned in some articles on etiology and in some articles on therapy and in texts, but the problem apparently has not been of enough interest or seeming magnitude to get much more than a passing glance from the experts who guide our thought.

Included in almost all classifications of the etiology of squint is the psychologic group. The size of this group, percentage-wise, varies with different observers and their particular interest in etiology.

Many authors include only a minute percentage of patients with psychogenic squints in their groups because a search for the exact cause is not made at all. In general, the great bulk of ophthalmologists are happy to distinguish between accommodative and nonaccommodative, and in the latter group to separate comitant from paralytic.

I hesitate to mention such a truism as the delicate balance of the binocular in the early years but it is just this precarious state that is responsible for deviations from minor causes. Perhaps there is no such thing as a minor cause. I'm sure a psychiatrist would not class psychic trauma as minor.

As we know, the interrelation of convergence, accommodation and vision, which are so strongly linked in childhood and remain so until middle-age, are very weakly related in infancy.

Of the three, convergence is the first to reach its highest level of develop-

ment. An infant can converge on an object at the age of 3 months, and at 6 months the convergence is all but fool-proof. At this same age (6 months), however, vision is estimated at 20/1000 and there is no ciliary muscle for accommodative purposes. So the conditions we always look for to control and maintain fusion are lacking to a great degree in the infant. The intermittent convergent state often seen in infants is the logical result of an attempt at forming a clearer image of a near object. The convergent state may also be seen in infants due to psychic factors; it is not uncommon to see a strong convergent movement before an infant bursts into tears.

While convergence appears early in infancy, it is, phylogenetically speaking, a rather recent innovation. Likewise, we are told that phylogenetically young functions are more susceptible to interference than others; it is no wonder that illness or physical or psychic trauma can cause permanent damage in the very young infant, for not only is an active convergence mechanism being stabilized, but the equally important convergence inhibitory mechanism is going through a refining process. And while I am about it, I might as well mention the divergence mechanism and its inhibitory mechanism, since these have become fashionable recently in the light of electromyographic tracings.

It must be quite easy to upset these very vulnerable functions by psychic trauma, just as walking and talking may be interfered with. While interference with binocular vision may be merely momentary, if repeated often enough it may lead to an irreversible state or deviation. Ophthalmologists don't think much about functional factors in the etiology of squint in the very young, but it might help if they did.

In the squints which develop at age 3 or 4, doctors are more apt to see

psychologic factors at work. The intermittent strabismus that becomes manifest when the child is mad, or embarrassed, or jumping with joy, is one of the most common types. It is true that the initiating factor may be a physical one, namely, fatigue, but they should be willing to recognize the several conditions that may operate to upset a heretofore well-functioning binocular mechanism. Many of these cases are hyperopic, and the abolition of overaccommodation by spectacles, cycloplegia, or the substitution of peripheral accommodation by miotics prevents the end effect on the binoculus from whatever cause. Perhaps this is the best they can do but surely a more exact cause might be found and corrected, rather than directing therapy toward preventing an effect.

Functional factors in causing a supposedly cured squint to recur are familiar. The following is a good example.

Case 1

The patient, a girl, was first seen at age 3 and had a constant, variable monocular esotropia. The onset of the constant deviation was 3 weeks before and the squint had first been noted as a definite, though intermittent, deviation about 6 months before she was examined. Hyperopia measured $+4$ prism diopters under atropine, so glasses were prescribed.

Under atropine cycloplegia there was a slight esophoria for both distance and near. Convergence and ductions were normal and there were no complicating vertical factors. A minimal amount of patching was applied and a minimal amount of orthoptic training was given to secure more stability with less correction. Just before her eighth birthday, examination showed vision to be 20/20 in each eye with and without correction. The eyes were steady under the cover test for distance and near with correction; an esophoria of 8 prism diopters for distance and 12 prism diopters for near was present without glasses. She was wearing slightly less than half her full correction because I try to remove glasses at age 9, if possible. For her eighth birthday she was given a present of a day at the beauty parlor. This included a permanent and she emerged from this with both her hair and her eyes curled. The squint, when the mother and child descended on me in

tears, was exactly like the squint of five years before. The angle was lessened by a full correction so $+2.00$ sphere fit-overs were ordered. Two weeks later the eyes were steady with full correction and fusion amplitudes were satisfactory. After another three months the fit-overs were removed, leaving a correction slightly less than half the total hyperopia.

There was no doubt in this particular case just what happened and when it happened and that remedial measures were both prompt and effective. I am sure there must be many cases that recur in a less dramatic manner, but from a definitely psychogenic or emotional upset. I am also sure that we could turn up these cases to some extent were we to take a more detailed history when the scene changes.

An entirely different functional factor in the treatment of strabismus is that of motivation. Now motivation is what they have to have, or be made to have, in order to improve or lessen a deviation, to learn physiologic diplopia by overcoming suppression, or any other phase of stepping up one rung of the binocular ladder. Technicians know only too well a patient's good days and his bad days. They know when there is some drive that will produce results and they know when that drive is lacking. They try to provide a background for the patient with pleasant surroundings and other trimmings. But orthoptics is a learning process, and learning takes concentration. Concentration usually comes only from a drive toward a desired goal. Even the fact that the goal is a desirable one is a psychologic concept. The patient has been surrounded with conversation that such and such an accomplishment is a desirable one. Since he really doesn't know what this goal will mean to him till he reaches it, he takes it on faith or, better, he has been brain-washed to believe this is so.

An example of motivation of a very high order is the following case.

Case 2

A 3-year-old had had an intermittent esotropia for three months which had become constant in the preceding ten days. Examination revealed the typical findings of an accommodative esotropia with the onset at age 3; a constant esotropia greater for near than for distance with bilateral overaction of the inferior obliques and overaction of both medial rectus muscles in the version tests. An atropine refraction disclosed a $+3.50$ error in each eye and glasses were prescribed. The vision was equal. Six weeks later a phoria was present with glasses for both distance and near, but an esotropia was present without glasses. There was normal fusion with good amplitudes for distance and near with glasses the following year. This happy situation continued for almost two years, then the child started to take lessons in twirling a baton with the idea of becoming a drum majorette. The baton used to hit the glasses with monotonous regularity and the child removed the glasses while practicing. At first there was diplopia and there were also tears. I told her she *could* hold her eyes straight. I did not believe that a child of 5 would work so hard for any goal, but I was wrong. In just three weeks a very proud mother and a very happy little girl showed me what motivation can do. She was able to hold her eyes steady during the cover test while accommodating for distance and near without glasses. This child is now 11, still has a hyperopia of 3.00 prism diopters, a slight esophoria with the Maddox rod, normal fusional amplitudes, normal vision without glasses, and several boy friends.

Whatever the difference of opinion may be regarding functional factors in the etiology of squint, there is practically unanimity of opinion in regard to the psychologic effect of a squint on the personality of the patient with a squint.³ As a matter of fact, the psychologic problem is actually or potentially greater than the ophthalmologic one. A child with a squint knows that he is different from his playmates, and differences, especially physical defects, cause derision. The child who is the target for jibes tends to withdraw from the group, first at play and, later, in school, and so develops a true inferiority complex. Even the patients of 10 years whom we think of as completely adjusted to their defect astound us by the

drastic change in personality postoperatively. And this change, as you all have noted, is in the direction of extroversion. Fortunately this condition does not go on for too long and elixir of phenobarbital will usually control the child and the mother.

The other type of child, the one who is an extrovert to begin with, may not let the squint get him down. Rather he tends to become more aggressive and destructive. He rises above his physical defect by his brashness and so lands in the group of incorrigibles. These patients should have surgical treatment as soon as an adequate diagnosis is made and should receive psychiatric treatment if the condition persists postoperatively.

While I am on the subject of surgical correction, it might be well to consider an operation for a case of esotropia that is largely, but not entirely, psychogenic. The chances of an overcorrection are very great here because the surgical correction itself may have psychotherapeutic value. This is not theory. Most of us are conservative in our surgical approach to cases of squints with variable deviations, and the functional group are usually variable. I'm really not trying to add more intangibles to the already complex preoperative evaluation, but now is a good time to think about such problems.

SUMMARY

1. Strabismus may be caused by a functional factor alone, and functional factors are often found in squints due to any cause.
2. The functional factor, if present, must be recognized as such and corrected. Patience and understanding are invaluable. Formal psychiatric help is rarely necessary.

3. Recurrence of a deviation in a supposedly cured case should arouse suspicions of a functional factor.

4. The effect of a squint on the personality of the patient may be so profound that the psychologic problem is greater than the ophthalmologic one.

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DIAGNOSIS OF VERTICAL MUSCLE PALSIES

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THIS discussion will be confined to the diagnosis of vertical muscle palsies with emphasis on the differential diagnosis between a superior oblique paresis and a superior rectus paresis. The latter is one of the most difficult and fascinating problems confronting us in trying to unravel the complex vertical muscle knot. It is found frequently if all muscle cases are carefully screened.

PHYSIOLOGIC BACKGROUND

The vertical recti make an angle of 23 degrees with an A-P axis of rotation in the primary position of gaze. Acting from this position the superior rectus is an elevator, adductor and intorter, and the inferior rectus is a depressor, adductor and extorter. The tendon of the superior oblique and the inferior oblique muscle subtend an angle of 45 degrees with the A-P axis in the primary position. Acting from this position the superior oblique is a depressor, abductor and intorter, and the inferior oblique is an elevator, abductor and extorter.

In the temporal field the vertical recti are reduced to elevators and depressors, while at the same time the obliques become chiefly intorters and extorters. Similarly, in the nasal field the obliques become elevators and depressors and the torsional function of the vertical recti is increased. In looking for underaction and overaction, therefore, the vertical recti are studied in the temporal field and the obliques in the nasal field,

the action of the muscle having been reduced to that of either simple elevation or depression.

A quick rule of thumb for remembering the cyclorotary actions of the vertical muscles is: the superior muscles are intorters, the inferior muscles are extorters.

The depressors of the eye are the superior obliques and the inferior recti. The elevators are the inferior obliques and the superior recti. The superior oblique and the contralateral inferior rectus are yoke muscles. The contralateral antagonist of a muscle is the direct antagonist of its yoke muscle.

To understand the principle of some of the tests discussed, a word about the static eye reflexes is considered necessary (fig. 1). As is well known, the

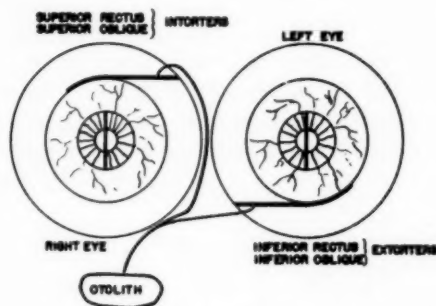


FIG. 1—Static reflex when head is tilted to the right shoulder.

ocular muscles are intimately related to the vestibular apparatus. The static reflexes send tonic impulses to the ocular muscles from the otolith apparatus in the vestibule by way of the eighth nerve

and the posterior longitudinal fasciculus. The static reflex is concerned chiefly with preventing the change in position of the eyes with change in position of the head with respect to gravity, i.e., to keep the eyes static. For example, when the head is tilted to the right with the eyes fixing straight ahead, the static reflexes tend to keep the vertical meridians of the corneas erect. The right eye is intorted by a balanced action of the superior oblique and the superior rectus, and the left eye is extorted by a balanced action of the inferior oblique and the inferior rectus. This is true torsion or "wheel action"; the eyes are neither elevated nor depressed.

DIAGNOSTIC AIDS

The following are considered valuable aids in making a differential diagnosis of vertical muscles:

1. History
2. Position of head
3. Prism and cover test (head tilt as well as cardinal positions)
4. Primary and secondary deviation
5. Red glass diplopia fields (linear rather than a point source of light is employed)
6. Lancaster red-green test (head tilt as well as cardinal positions)
7. Phorometer (measurement of the cyclotropia as well as the hypertropic component for near and distance)
8. Bielschowsky's sign
9. Helmholtz head tilt test (Stine modification)

History

A history of trauma may be helpful, particularly if there has been damage to the trochlear region by accidental injury or an operation on a sinus. In some children, history of how the head is held in everyday life is important. Due

to preconditioning by parents, the child may hold his head erect in the office when there is normally a head turning or tilting. In the adult case, a history of diplopia and what the patient does to relieve it is most helpful. If it is worse at distance, a vertical rectus is suggested. If more pronounced at near, an oblique may be at fault; and if reading produces the greatest difficulty, a superior oblique is under suspicion. If the vertical diplopia is relieved by lowering the head, a depressor is involved; an elevator, if the head is thrown back to obtain comfort.

Position of Head

The position of the head is most valuable in the diagnosis of a superior oblique palsy. Torsional diplopia is very annoying and can be relieved only by head tilting. Head tilting to the opposite shoulder is common, particularly in children, when a superior oblique is involved (fig. 2). Since the



FIG. 2—Head tilting to left shoulder in patient with paresis of right superior oblique.

superior oblique is an intorter, the vertical meridian of the cornea is extorted. By tilting the head to the opposite shoulder this meridian is brought to the vertical, and the static reflex adjusts the vertical corneal meridian of the sound eye so that the two meridians are parallel and binocularity is possible. Tilting the head to the opposite shoulder in superior oblique palsy also tends to relieve not only the torsional but also the vertical diplopia.

I have seen several cases of superior oblique paralysis in which the head was tilted to the same side, apparently to effect as wide a separation of the images as possible.

Occasionally a patient with superior oblique paralysis will turn the head to the good side so that the involved muscle is acting in the temporal field. Its depressing action is then reduced to a minimum and the vertical diplopia is greatly relieved. The chin is rarely depressed in a superior oblique palsy.

Head tilting is exceedingly rare in the case of superior rectus paralysis. Elevation of the chin is more common, not only to get the eyes out of the field of action of the involved muscle but occasionally because of an associated ptosis.

Abnormal positions of the head are rarely encountered in palsies of the inferior rectus and inferior oblique.

Prism and Cover Test

The prism and cover test may be helpful when done in the cardinal positions. However, as every ophthalmologist and orthoptist knows, many patients cooperate poorly when trying to neutralize the recovery motion in the oblique positions of gaze. Prism measurement for near and distance must not be overlooked. If the hypertropia is greater for distance, a vertical rectus may be at fault. If greater for near, an oblique is usually implicated.

The importance of doing prism and cover measurements in the head tilt positions cannot be overemphasized, particularly in children. In superior oblique palsy the hypertropia will be much greater when the head is tilted to the affected side and much less when tilted to the opposite side.

Primary and Secondary Deviation

It goes without saying that particular attention should be given to the difference in the degree of vertical deviation when first one and then the other eye is fixating. The deviation is greatest when the paretic eye is fixating (secondary deviation) and less pronounced when the sound eye is fixating (primary deviation). The cover, red glass and Lancaster red-green tests all help to determine the primary and secondary deviations. I concur with the view expressed by Bielschowsky, however, that in many long-standing squints, due probably to contracture of the direct antagonist, the originally paretic muscle cannot be discovered except by history. The squint becomes quite comitant.

Red Glass Diplopia Fields

Red glass diplopia fields are helpful in the usual case. The greatest separation occurs in the field of action of the paretic muscle, and the more peripheral image is the image of the paralyzed eye. A linear light is preferable to a point source of light. This permits the determination of the torsional as well as the vertical component. In the case of a left superior oblique weakness, for example, the greatest relative intorsion of the images will occur on down and left gaze, the greatest vertical separation on down and right gaze. With a cooperative patient, primary and secondary deviations can usually readily be determined.

There are a number of cases, however, in which the diplopia field is of

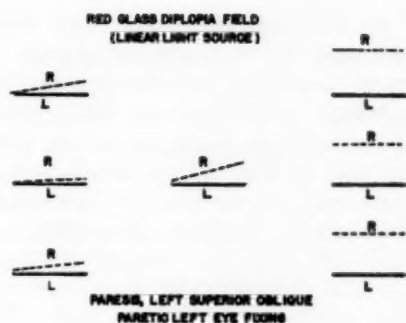


FIGURE 3.

little or no value. This is especially true in long-standing squints. Also, when the paretic eye is the fixing eye, the diplopia field may indicate two muscles, the paralyzed muscle and its underacting contralateral antagonist (inhibitional palsy of Chavasse) (fig. 3).

Lancaster Red-Green Test

The Lancaster red-green test is considered preferable to the red glass test except for the high cost of the instrument. It is much more accurate, is accomplished with minimal exasperation, and is more reliable in older children. The separation of the images should not only be recorded in the cardinal positions of gaze but also with the head tilted to each shoulder, the eyes being directed to the center of the screen. The vertical separation of the images, for example, will be much

greater when the head is tilted to the paretic side in the case of a superior oblique paralysis (fig. 4).

Phorometer

The phorometer is particularly valuable in measuring small vertical deviations. It accurately records the difference, if any, in the hypertropia at near and distance. It is also quite handy for measuring the degrees of cyclotropia present. A Maddox rod is placed before each eye with the light streaks horizontal. If the linear images are not sufficiently separated, a small vertical prism is introduced. The patient is asked to adjust the tilted light streak until both streaks appear to be parallel. The amount of cyclotropia in arc degrees, positive or negative, is then read off the scale on the Maddox rod directly. In positive cyclotropia the globe is extorted; in negative cyclotropia it is intorted.

Bielschowsky's Sign

Bielschowsky's sign in superior oblique palsy is practically pathognomonic. The involved eye moves upward as the head is tilted to the paretic side. For example, a left hypertropia due to a paretic left superior oblique becomes much worse as the head is tilted to the left shoulder (fig. 5). This sign is often not obvious, but can usually be detected with the

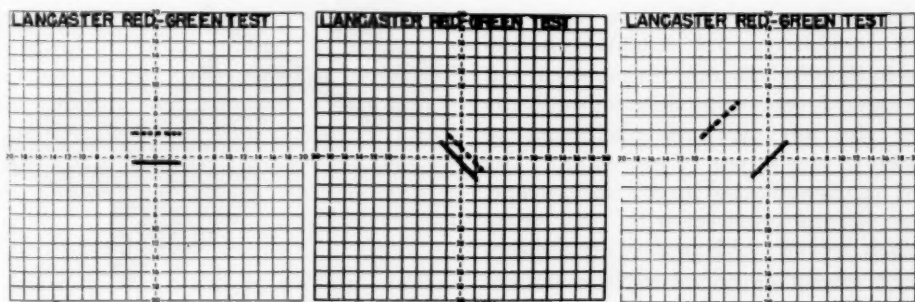


FIG. 4—Findings for patient with paresis of left superior oblique, right fixing. [— (Right Eye); --- (Left Eye)] (Left) Head erect. (Middle) Right head tilt. (Right) Left head tilt.



FIG. 5—Positive Bielschowsky's sign in patient with paresis of left superior oblique.

cover test, re-emphasizing the desirability of doing the cover test in the head tilt positions.

Helmholtz Head Tilt Test

The Helmholtz head tilt test is of tremendous value in diagnosing a superior oblique weakness, particularly in differentiating it from the contralateral superior rectus. The Stine modification using a Brewster-type hand stereoscope is employed (fig. 6). A horizontal red line is drawn on a plain white card so that it will be seen only with the right eye. A similar horizontal line in blue is drawn on the same card at the same level so that it is viewed only with the left eye. A circle can be drawn in the center of each line. The card is then placed in the stereoscope. The normal person will fuse these images in all positions of the head. Again taking as an example a patient with left superior oblique paresis with the normal right eye fixing, the findings

are as follows: with the head erect there will be a moderate degree of vertical separation of the images, the blue image of the left eye being lower and intorted; on head tilt to the right there will be less vertical displacement and intorsion of the blue left eye image, or fusion will result; on tilting the head to the left both the vertical displacement and the intorsion of the left eye image will be markedly increased. The patient is asked to draw the images as seen immediately following each position of the head with no prompting. The images in the case of a right superior rectus palsy will show greater vertical separation and relative intorsion on head tilt to the right shoulder and will be fused or much closer together on tilting the head to the left (fig. 7). The value of this test cannot be overemphasized. It is frequently the clincher in an otherwise difficult differential diagnosis.

The greater vertical separation of the eyes in superior oblique palsy on tilting the head to the paretic side (Bielschowsky's sign, cover, Lancaster red-green and Helmholtz tests) is explained in the following manner, again using a left superior oblique as an example: on tilting the head to the left shoulder, the left superior oblique and

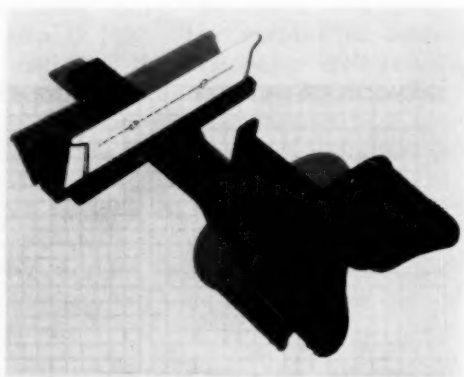


FIG. 6—Equipment necessary for Stine modification of Helmholtz head tilt test.

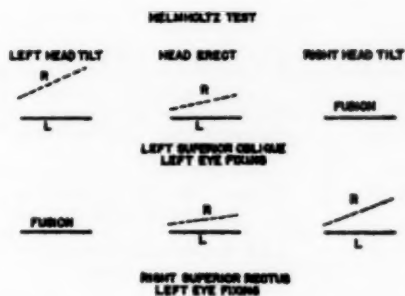


FIGURE 7.

the left superior rectus receive a reflex innervation from the otolith apparatus to intort the left eye as explained above. Since the elevating action of the normally functioning left superior rectus is unopposed by the depressing action of the underacting left superior oblique, the left eye deviates upward.

DIFFERENTIAL DIAGNOSIS BETWEEN A SUPERIOR OBLIQUE AND A CONTRALATERAL SUPERIOR RECTUS PARESIS

When the paretic eye is the fixating eye there is not only underaction of the paralyzed muscle but also of its contralateral antagonist. Chavasse called this inhibitional palsy, but Adler gave the correct explanation when he pointed out that this was merely underaction of the contralateral antagonist based on Hering's law of equal innervation to both eyes.

Continuing with the left superior oblique as an example, this time with the paretic left eye fixing: there will be a left hypertropia in the primary position which decreases or disappears in all positions of left gaze; on looking down and to the right the left hypertropia will be greatly increased because of the left superior oblique palsy; and on looking up and to the right the left hypertropia will also increase due to the underaction of the right superior rectus, the contralateral antagonist. This creates a major problem in the differential diagnosis of

the two muscles. Which is the paretic and which is the underacting muscle? Are both muscles actually paretic?

Cover, red glass and Lancaster red-green tests are frequently of little or no value when done in the usual cardinal positions of gaze. To recapitulate the important points in the above discussion, the differential diagnosis can usually be made as follows:

1. Position of Head

Left superior oblique: Tilts head to right shoulder or turns head to right side.

Right superior rectus: The chin is elevated, or if tilting occurs, it will be to the left shoulder.

2. Bielschowsky's Sign

Left superior oblique: On tilting the head to the left (same side) the left eye will be seen to move upward.

Right superior rectus: On tilting the head to the right (same side) the right eye will show no movement or a slight downward movement. The Bielschowsky sign is absent.

3. Prism and Cover Measurements in the Head Tilt Positions

The findings described under Bielschowsky's sign may be brought to light by these measurements when they are not grossly apparent.

4. Lancaster Red-Green Test

The findings in the head tilt positions corroborate the prism and cover measurements.

5. Helmholtz Head Tilt Test

Left superior oblique: There is greater vertical separation and relative intorsion of the images on tilting of head to the left, less vertical separation and intorsion on tilting head to the right.

TABLE I
DIFFERENTIAL DIAGNOSIS

	LEFT SUPERIOR OBLIQUE	RIGHT SUPERIOR RECTUS
POSITION OF HEAD	Head tilts to right shoulder or turns to right side	Elevates chin or tilts head to left shoulder
BIELSCHOWSKY'S SIGN	Present on tilting head to left shoulder	Absent on tilting head to left or right shoulder
PRISM AND COVER TEST	Left hypertropia greater in left head tilt position	Left hypertropia greater in right head tilt position
LANCASTER RED-GREEN TEST	Left hypertropia greater in left head tilt position	Left hypertropia greater in right head tilt position
HELMHOLTZ TEST	Greater vertical separation and relative intorsion of images in left head tilt position	Greater vertical separation and relative intorsion of images in right head tilt position

Right superior rectus: The vertically separated and relatively intorted images will decrease on head tilt to the left and increase on tilting to the right. Changes in the position of the images are less dramatic in the case of a superior rectus palsy (table I).

SUMMARY

It is imperative to have a good understanding of the physiology of the extraocular muscles before an accurate diagnosis of the palsies can be made. This is especially true in the case of the more

complex vertical muscles.

Emphasis has been placed on the diagnosis of superior oblique pareses, with special attention being given the differential diagnosis of a superior oblique on one side and a contralateral superior rectus palsy. Head tilting, Bielschowsky's sign, cover and prism measurements in the head tilt positions and the Stine modification of the Helmholtz head tilt test are the cardinal procedures to be employed in making this frequently difficult differential diagnosis.

ORTHOPTIC TREATMENT OF PATIENTS WITH UNFAVORABLE PROGNoses

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THE compilation of statistics from any large field of endeavor gives information of a kind, but lacks the interest of individual analysis. Since the field of strabismus is large, detailed treatment of individual cases is seldom possible and reported treatment must, of necessity, be given in percentages most of the time. Generally, the patients with squint who receive orthoptic training are classified into favorable or unfavorable groups, but usually the report does not include individual therapy. Favorable and unfavorable prognoses probably vary considerably with different schools of thought, and it is not in the scope of this paper to discuss them. Rather, I will present a complete case history of one patient given an unfavorable prognosis.

This presentation states types of corrective procedures used and the patient is exceptional, but not unusual, in our unfavorable classification. This does not imply that all patients with poor prognoses give as satisfying results as this one. However, over 50 per cent of our patients with unfavorable prognoses have achieved results equal to successful corrections of patients with favorable diagnoses; that is, single binocular vision for both near and distance. Over 35 per cent have some binocular function, and less than 15 per cent had purely cosmetic corrections.

I will define the binocular classifications. Single binocular vision is considered present if first and second grades of foveal binocular vision are maintained for near and distance without the occurrence of suppression or diplopia during normal use of the eyes. Stereopsis may or may not be present. Binocular vision simply denotes that both eyes are used simultaneously. Peripheral fusion may be present with either foveal suppression or diplopia. Bifoveal reading is not present and fusional amplitudes are limited. Usually, there is a deviation present which invites suppression amblyopia, or abnormal correspondence, and often an increase in the deviation. These factors preclude the possibility of a normal functional development. A patient should have every opportunity to develop a normal single binocular pattern.

The time element is unimportant when weighed against the successful development of bifoveal fusion. The importance of bifoveal fixation practice cannot be overemphasized in orthoptic training. Patients with unfavorable prognoses sometimes respond favorably to bifoveal stimulation. Bifoveal stimulation and eradication of suppression cannot be left to surgical procedures alone; even a successful operation usually leaves a residual deviation large enough to prevent bifoveal fusion. This is especially noted in patients who have had alternating fixation since birth, patients with deep suppression and anom-

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alous correspondence, where patterns of seeing have already been learned. Bifoveal fixation is not present in refractive corrections which seemingly render the eyes parallel but leave a residual deviation. This prevents foveal visual development and the possibility of bifoveal fusion.

Operations performed on the eye muscles of a child may change a favorable prognosis to an unfavorable one. This change occurs because the operation masks an undeveloped bifoveal fixation in a successful cosmetic correction, or one where only peripheral fusion develops in an undercorrection or overcorrection. Further unfavorable prognoses may develop when early, frequent and radical surgical procedures have been performed.

In cases where a partial overlapping of the single binocular fields occurs, and partial fusion is sometimes present, bifoveal fusion is not present and an unfavorable prognosis results. This situation becomes favorable when bifoveal fixation is acquired through the training which allows foveal fusion and binocular stability to develop. When deep suppression and/or incomitance are present, foveal fixation practice with first one eye, then the other, fixating the smaller of the two first grade, foveal macular targets is of paramount importance.

In treating a patient with strong suppression the first grade, foveal macular targets are necessary, though less conducive to binocular function. This is especially true in the case of a long duration of squint. Once spontaneous diplopia is present and a bifoveal fixation pattern is established, the use of second grade targets is desirable. Infants with occlusion have very little suppression when they reach the age for instrument training, and, therefore, have the advantage of using second grade macular

targets sooner than patients with deep suppression. Second grade macular targets help the patient to fuse when varying lateral and vertical deviations are present. Unless a bifoveal fixation pattern has been established, fusion may be erroneously reported subjectively when, actually, a slight tropia will be found when the patient is tested with foveal targets.

In presenting the following case some repetition was unavoidable in order to give the pertinent facts regarding the detailed corrective procedures.

CASE REPORT

A. K. was first seen in February, 1947, at age 15 months. Since birth she had had crossed eyes and a right head tilt.

Examination showed:

Alternating fixation, slight preference left eye, bilateral nystagmus. Left eye fixating, nystagmus slight. Right eye fixating, nystagmus marked. Head tilt marked to right side.

Rotations—Lag-RLR, marked lag-LLR, LSR

Versions—Overaction, inferior obliques, RSO, RMR

Deviation— 75Δ Et' — 65Δ Et, alternating sursumduction

Fundus—Negative

Cycloplegic—R Plano $+50 \times 90$, L Plano $+1.00 \times 120$.

Refractive correction not given

Visual Acuity—Not ascertained

Diagnosis:

Noncomitant alternating nonaccommodative convergent strabismus—Innervational verticals

Prognosis:

Unfavorable

Therapy:

From 1947 to 1952—Complete occlusion, depending upon fixating eye. Re-examination every 8 weeks (fig. 1).

Daily preoperative training on major amblyoscope—age 7 (fig. 2):

Visual Acuity—SC R 20/40, L 20/40+1

SC Major Amblyoscope—Subjectively, not able to locate, constant variation in vertical and lateral deviations. Objectively, SMP not present, bifoveal fixation practice, objective angle of deviation 55Δ to 80Δ



FIG. 1—Case A. K., age 6. Preoperative 1951. Alternating esotropia. Paresis of the left superior rectus and the left lateral rectus. Alternating sursumduction.

Et', 50Δ to 60Δ Et. 4Δ to 16Δ right alternating sursumduction.

Surgical correction:

August, 1952—2 mm. recession RMR, 3 mm. resection LRL.

Occlusion therapy discontinued. To return 1 month later for re-evaluation.

Postoperative examination—Head tilt present (fig. 3)

One month postoperative—September 1952:

Major Amblyoscope—SC Subjective—variable, not able to locate. Objective—SMP not present $+26\Delta$ to $+35\Delta$ Et', $+26\Delta$ to 30Δ Et. Alternating sursumduction varying from left to right, zero position at times.

Visual Acuity—SC R 20/40, L 20/40.

Right eye preferred.

Return to complete occlusion right eye depending upon fixating eye. Return 1 month. Orthoptic training once a month for 2 days from 1952 to 1955.

Surgical correction:

August, 1955—2 mm. recession LMR, 4 mm. resection LLR.

Occlusion therapy discontinued. To return 1 month for re-evaluation and orthoptics.

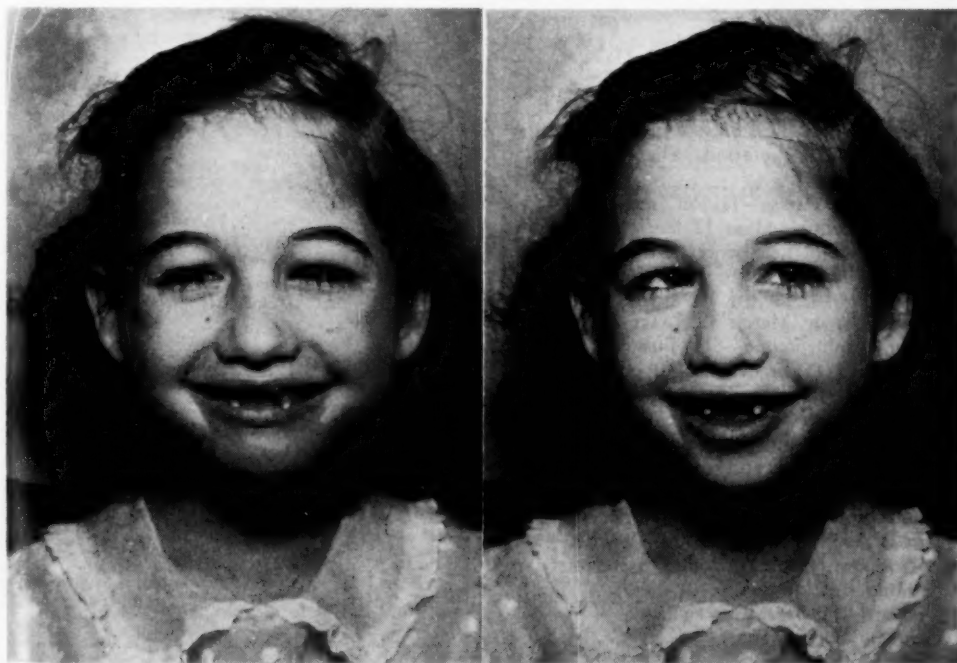


FIG. 2—Case A. K., age 7. Preoperative. Spontaneous fixation either eye. Slight head tilt.



FIG. 3—Case A. K., age 7. One month postoperative. Occlusion therapy discontinued postoperatively. Head tilt returned.

One month postoperative—September 1955 (fig. 4):

Major Amblyoscope—SC SFP 11Δ to 17Δ Et', 4Δ to 12Δ Et.

Slight left alternating sursumduction 0 to 3Δ . No suppression.

Right eye still preferred. Slight diplopia on bar separator.

Recession exercise—Fusion red and white lights maintained to 12 feet. Occlusion of right eye for near work. Orthoptics once a month for 2 days to March 1956.

Re-evaluation on March 1956, age 11

Major Amblyoscope—SC Fusion 15Δ Et', Fusion 12Δ Et, Vertical 0 position

For near—Fusion maintained to -7Δ with diverging and to $+22\Delta$ with converging

For distance—Fusion maintained to -5Δ with diverging and to $+16\Delta$ with converging

Slight diplopia on bar separator

Visual Acuity—SC R 20/50+, L 20/50+

Rotations complete—slight nystagmus extreme rotations

Versions—no overaction or lags

Head tilt not present

Cycloplegic—R $-25 +1.00 \times 90$, 20/50+; L $+1.00 +.37 \times 135$, 20/50+

Refractive correction not given

Continue partial occlusion right eye and orthoptic training

Intensive orthoptic training—4 days, May 1956

Major Amblyoscope—SC SFP $+32\Delta$ to $+35\Delta$ Et', $+30\Delta$ to $+32\Delta$ Et

First day—Vertical 0 position

Second day—SFP $+25\Delta$ to $+22\Delta$ Et', $+23\Delta$ to $+20\Delta$ Et

Third day—SFP $+20\Delta$ to $+22\Delta$ Et', $+15\Delta$ to $+12\Delta$ Et

Fourth day—Fusion $+16\Delta$ Et', $+12\Delta$ Et—Vertical 0 position

For near—Fusion maintained to $+13\Delta$ with diverging and to $+30\Delta$ with converging

For distance—Fusion maintained to $+9\Delta$ with diverging and to $+22\Delta$ with converging

Slight diplopia on bar separator

Continue partial occlusion right eye

Orthoptic training—4 weeks, July 1956

Major Amblyoscope

First day—SC SFP $+25\Delta$ Et', $+15\Delta$ Et

High—SFP $+28\Delta$ Et', $+20\Delta$ Et

Low—SFP $+30\Delta$ Et', $+22\Delta$ Et

Variations—SFP $+34\Delta$ Et', $+22\Delta$ Et

Last day—SFP $+5\Delta$ Et', $+5\Delta$ Et

Varying left to right alternating sursumduction

0 position maintained more often than not



FIG. 4—Case A. K., age 10. One month postoperative. Partial occlusion therapy for near work.

Fusion $+5\Delta$ Et', Fusion $+5\Delta$ Et

For near—Fusion maintained to $+1\Delta$ with diverging and to $+24\Delta$ with converging

For distance—Fusion maintained to $+2\Delta$ with diverging and to $+20\Delta$ with converging

Vertical 0 position

Occasional fusion with foveal targets on bar separator

Recession exercise to 16 inches and with 4Δ base out to 20 feet

N.P.C. 2 to 3 inches

Visual Acuity—SC R 20/40, L 20/40

Continue partial occlusion right eye for near work only

Orthoptic training—1 day, November 1956

Major Amblyoscope—SC SFP $+3\Delta$ Et', $+1\Delta$ Et

Vertical 0 position

Off instrument—hand Prisms

Vergence—33 cm. D' 4Δ , C' 22Δ

Fusion— 2Δ base out, 20 feet

N.P.C.—1 to 2 inches

Continue partial occlusion right eye for near work only

Orthoptic training—1 day, January 1957

Major Amblyoscope—SC fusion 1 mm., foveal targets $+4\Delta$ Et', $+2\Delta$ Et

For near—Fusion maintained to 1Δ with diverging and to $+14\Delta$ converging

For distance—Fusion maintained to 0 with diverging and to $+10\Delta$ with converging

Vertical 0 position

Fusion with foveal targets maintained longer on bar separator

Continue partial occlusion right eye for near work

Orthoptic training—1 day, March 1957—age 12 (fig. 5)

Major Amblyoscope—SC fusion $+3\Delta$ Et', fusion $+2\Delta$ Et

Vertical 0 position

For near—Fusion maintained to -1Δ to -6Δ with diverging, to $+35\Delta$ with converging

For distance—Fusion maintained to -1Δ to -2Δ with diverging, to $+25\Delta$ with converging

Recession exercise to 14 feet and base out 2Δ to 20 feet

Four dot test—4 dots at 10 feet and 5 at 20 feet

Cheiroscope—Figures slightly irregular and slightly convergent

Bar separator—Foveal fusion primary position—reading 20/100 print. Fusion maintained three letters on each side of center fixation with side movements.

Continue partial occlusion right eye for near work

Remarks:

Continued improvement expected. Office orthoptic training probably for another year at 1 month intervals with emphasis on daily bar reading at home along with vergence exercises.

Prognosis:

Favorable for 20/40 single binocular reading by next year.

Note: It takes longer to achieve the first single binocular reading efforts than the final, if bifoveal fusion is maintained. Once bifoveal fusion is maintained and binocular accommodation begins to function, improvement continues through the use of this act. Unless this function is well established, alternating monocular reading becomes the pattern. This fact was manifest on rechecking patients who had not received orthoptic training for five years or longer. Near binocular visual acuity is not comparable to distance visual acuity due to the increase in accommodation for near on the bar separator.

SUMMARY

As this patient lives over two hundred miles from Atlanta, and weekly office visits were impractical, visits had to be as few as possible. Visits every six to eight weeks were sufficient for office observation and direction of home occlusion during preschool years. At this time occlusion therapy was complete and constant. Occluding the fixating eye was determined each day after an hour's observation of both eyes by the parent.

This therapy, though largely negative, is nevertheless an important part of the squint correction. By instituting occlusion before two years of age, stimulation and direction of the reflex development is possible. Undesirable factors such as amblyopia, anomalous retinal correspondence, false projection and suppression are prevented. These are not organic conditions, but learned quantities, and all, except false projection, stem from binocular functions.

False projection could often be included in the above list when found in



FIG. 5—Case A. K., age 12. Versions complete. Head tilt not present.



FIGURE 5 (continued)

large deviations with a marked paresis. A marked paresis is often found in the early stages. By the use of occlusion therapy, binocular associations in these cases are eliminated, and monocular development toward the normal is possible. In addition to the sensory aspects, the motor apparatus receives helpful stimuli from occlusion therapy. The deviated eye, through occlusion therapy, becomes the fixating eye and tends to stimulate excursion of the muscles in that eye. With the development of foveal vision and the absence of suppression and anomalous correspondence, the patient has only the fusion problem to combat when old enough for fusion training. Occlusion therapy of some form is necessarily continued, to prevent foveal suppression, until such time as the obstacles to normal single binocular vision are eliminated.

I have a rule I usually use which goes like this, "As long as an esotropia exists, occlusion persists." Various types of

occlusion, from complete to partial, were used during the treatment period of the above patient. The use of occlusion is necessary in the majority of cases, as it is hardly possible to expect a patient not to suppress when a deviation, however small, prevents comfortable binocular use of the eyes. The above statement excludes those patients who have a constant diplopia as a result of surgical procedures, trauma or other such mechanical causes. The patient's symptoms and appearance are not the only indications for occlusion, as the patient may be comfortable and have good cosmetic results, but still have suppressed foveal vision. Binocular instrument training begins with the patient as soon as conditions indicate. These conditions are governed by acuity, age and stability.

Treatments of this patient were four to eight weeks apart, for one to two day periods during the school year, with two to four weeks intensive daily train-

ing during summer vacations. The learning carry-over from one visit to another makes orthoptic training possible for patients seen infrequently. Instrument training was not begun until the patient was seven years old because of low visual acuity and instability. Until a certain amount of stability has developed, attempting to train binocular coordinations is futile. The age of the child alone does not determine stability. Some children are able to begin training as early as five years and others not until eight years. Certain psychologic factors must be considered before beginning instrument training. The child who matures slowly cannot begin orthoptic training as soon as others. Such a child will not accept the temporary discomfort of diplopia and the effort and exactness of bifoveal fixation. To avoid this situation, the deviated eye will be turned further in where comfortable monocular fixation can be maintained. This can be demonstrated in cases where the deviation is small enough to excite diplopia and the fusional range is insufficient for single binocular vision.

The bar separator and other devices of this type combat this type of suppression by eliciting bifoveal fixation. Once a child has matured sufficiently to accept diplopia and work with it, rather than avoid it, the length of time for correction is greatly reduced. Also, as long as the child remains too immature to accept the diplopia stage, monocular fixation continues to be present. Monocular vision may be caused by suppression while the eyes remain in parallel alignment, or by turning the nonfixating eye in as far as necessary to avoid foveal fixation and diplopia. Monocular vision occurs in patients surgically treated, as well as in those not surgically treated, in varying degrees of deviations. The technician can give valuable information to the ophthalmologist regarding the

child's progress in accepting orthoptic training responsibilities. The patient's progress is one of the indications to consider in planning surgical correction.

Some surgical corrections are successful for patients under five years old and others are not. If an operation is unsuccessful to such an extent that the deviation is scarcely changed, further immediate surgical correction usually will be unsuccessful again. Surgical correction in later years often results in overcorrection. Procedures used to correct squint do not depend solely on the maturity of the child. But time must be considered in order to work out a functional correction for the child that will be advantageous in adult life.

Next in importance to the diagnosis are the treatment and methods used for correction. Orthoptic technicians know the dual role targets play in their work. For the child, the targets used are just so many pictures. For the technician, the targets are selected to reveal and correct a given condition. A direct means of establishing a binocular relationship was necessary for the patient discussed who had had an alternating esotropia since birth. Macular targets were selected and used in the beginning of treatment so that an objective control of foveal fixation could be maintained and a normal relationship established. Also macular targets were used to aid and confine the movements of the constantly varying deviation. After a normal relationship developed, peripheral and the more exacting foveal targets were added. Second grade macular targets for patients with alternating sursumduction are beneficial if bifoveal fixation is steady and suppression does not exist with first grade foveal macular targets. In using second grade targets, an alertness to suppression and foveal fixation is necessary, especially in the beginning of treatment.

CONSERVING VISION

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How important is sight? About 4,000 years ago in the Babylonian code (usually called the code of Hammurabi) we find proof of the high value the Babylonian people placed on the eye. The king did not mince words when he wrote:

The physician who opens an abscess in the eye of a man with a bronze lancet and saves that man's eye, he shall receive 10 shekels of silver as his fee. If a Freeman, 6 shekels, and if a Slave man, 2 shekels from the owner to the physician. But if a physician operates on a man and cause the man's death; or lance an abscess on the eye and destroys the man's eye, they shall cut off his fingers. If it is only a slave's life or eye, the physician must pay half the slave's market value to the owner.

Not only is the code illuminating as a commentary on the social order of the day, but it also shows clearly that in those ancient times the eye and life were held in equal esteem.

To many of us, sight is priceless. Although we may not have realized how intimately eyes are bound up with all man's waking life and actions, it is certain that vision is the most cherished of his possessions, next to life itself. Some are even more positive and consider life without vision valueless.

We live by sight. Of the five senses, vision alone is responsible for more than 80 per cent of our awareness of the world around us. A sharp noise, an unusual smell, a strange touch, and immediately our eyes look for the source. We must see.

The human is endowed with the finest visual apparatus in the animal world. The eye is far more efficient, complex, adjustable and accommodating than any instrument designed by man. Like all good machinery, it does many things that make life enjoyable. But, just as machines must be adjusted, must be cared for, must be repaired, so it is with our eyes.

But let us not labor the point. All of us are aware of the importance of our eyes and do not have to be sold on the proposition. Sadly though, many of us appear to take our wonderful eyes for granted while they are working well. But when our eyes are not functioning properly, immediate care should be taken to correct the trouble. However, this does not usually happen. It is human nature to procrastinate, and unfortunately the delay may lead to sight loss. This in essence is the reason for our program—to prevent this loss.

Prevention of blindness is both an old and a new responsibility in public health. Around the turn of the century the control of ophthalmia neonatorum by means of postnatal prophylaxis became a mandatory, at least a regulatory, function of health departments. At the same time health agencies began to participate in school vision programs. Thus organized vision conservation had its beginning in public health.

More recently, however, our horizons have broadened, and the total problem of visual incapacity has presented itself as a public health responsibility. W. K. Kellogg Foundation grants were made to

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Oregon in 1950 for programs in prevention of blindness and conservation of vision.

The main purpose of the Oregon program is to determine how a vision conservation program could be strengthened. In formulating the objectives of such a program, it was necessary to examine the various sight-saving conditions in terms of their amenability to control.

Oregon's program has four major objectives:

1. To prevent the occurrence of blinding disease
2. To halt the progression of pre-existing disease toward blindness
3. To restore lost vision
4. To minimize the handicapping effects of visual impairment

You will notice that our objectives are simply preventive. They are not diagnostic or prognostic. Preventive action is the theme of our program.

Blindness, like other disabling conditions, can in a large measure be prevented, and valuable vision can be preserved much as we preserve other natural resources. But blindness has many different causes, therefore prevention must take many different approaches. In Oregon, we found it necessary to look critically at our already blinded population, and thereby to try to determine some of the special program areas of need and practicability. Since reporting of blindness is not mandatory in Oregon, a vision register of the visually handicapped was established to determine the leading causes of blindness worthy of program emphasis. In the analysis of our register we discovered the leading causes of sight loss in children to be accidents, ophthalmia of the newborn, and retrolental fibroplasia. But legislation and regulations based upon research and study have provided primary prevention, i.e., action to pre-

vent blindness even before the cause begins.

Other diseases found to produce loss of sight, particularly in the middle and later age groups, were glaucoma, diabetes, and arteriosclerosis. Many of these cannot be cured in the light of our present knowledge, and the best we can hope to do is retard some conditions, such as glaucoma and diabetes. This kind of prevention is coming to be known as secondary prevention. The disease is clearly present, but through early detection and proper treatment, the extent of disability is lessened or further loss of sight is prevented. In some instances such as cataracts, retinal detachment, and corneal disease, surgical treatment often restores sight if performed at the proper time.

Viewed in terms of total potential years of blindness, however, and in relation to the social, the emotional, and the economic results of long-term visual incapacity, we have felt that an over-all program in vision conservation must cross all age groups and all etiologies. To this end considerable attention is given in the Oregon program to the case finding, referral, and follow-up of children's eye conditions, in which the number of potentially blinding eye conditions is small and the total of less serious, but readily correctible defects, large. This leads to the third method of prevention. Prevention by educational processes and by disseminating knowledge about symptoms of eye conditions of the child and the importance of seeking and carrying out competent professional advice. Its objective is to provide nurses (school and public health), teachers, volunteers, parents, professional people, and others who work with children an opportunity to obtain up-to-date knowledge about screening procedures for referrals and follow-up on visual defects, eye health, and social adjustment of the visually handicapped child.

The theory of vision screening is simply the separation of the apparently normal from the probably abnormal. Screening is necessary because many parents neglect responsibility for securing good eye care for their children or are unaware of the need. Screening is not intended to supplant complete definitive examination. It is simply a means of determining which children are in need of complete examination. The practicability of all children receiving complete examination is questionable, both numerically and economically, and besides parents are often suspicious of the motives of professional groups. By screening, detection, and referral for correction of vision defects, many youngsters can be saved from needless social, emotional, and educational maladjustments. Therefore screening does have a basic and useful role in vision conservation.

A preschool or kindergarten vision screening program, in addition to detecting common refractive errors, assures observation of the child for signs of other eye trouble, particularly those defects, such as muscle imbalance and suppression, which may cause a visual loss if neglected. This vision screening program also familiarizes the child with the procedure so that by the time he is admitted to school, he can be tested again with greater ease and reliability.

In the discussion of muscle imbalance and refractive errors in their relationship to vision screening, research indicates that by using good screening methods and early examination, four million people might be saved from losing 50 per cent or more of their sight. It is the confirmed belief of vision conservation people—medical, professional or otherwise—that all children, before age six, should be screened and examined for eye defects which, without proper correction and care, may lead to loss of sight. Of all muscle balance and sup-

pression problems, the majority occur long before the child comes to school. Therefore the early testing of these children will carry much significance.

Vision screening of school children, while widely accepted in principle, has long been a matter of debate in terms of methodology and technique. Much has been written and many studies have been conducted in an attempt to select a single simple and efficient device whereby children needing eye care may be picked from their classmates.

In Oregon we feel that parent-teacher-nurse observation is of key importance in vision screening. Coupled with this observation is Snellen testing. The Snellen E chart is used in the primary grades. In the intermediate and upper grades the Snellen alphabet chart is commonly used. National studies agree that the Snellen test plus observation is 80 to 85 per cent effective. This is very high for a screening method. It rates high also on a time and money basis; as it produces only about 15 per cent of needless referrals.

Even so, there has been a move in the schools toward the use of mechanical and stereoscopic devices in the screening procedure. From recent studies conducted with these instruments, the degree of accuracy in general does not equal that of the Snellen method, thereby resulting in a high percentage of needless referrals. This is regrettable, because a high rate of needless referrals rapidly brings vision screening programs into disrepute among parents, causing them in many cases to ignore the referral notices. Therefore, use of instruments has not been recommended in school programs. However, it should be stated that although these devices do have limitations, they also have some advantages which could aid in detecting certain eye problems needing care. No single screening device available today is completely adequate. Through ex-

perimentation and research it is hoped that an efficient accurate and simple instrument giving standardized results for accurate referrals may be forthcoming.

The professions concerned with eye care, treatment, and correction all have a place in vision conservation. The first responsibility of these professions is to conserve, restore, and make useful as much vision as possible for each client or patient they serve. Vision is not something we are born with, but something that we develop by process of conditioned reflexes. If the development is irregular, the resulting factor is abnormality of the eye. Early detection through screening, case findings, referrals, and examination requires cooperation and communication among all of us in vision conservation. Follow-up regarding proper treatment for eye conditions is a continuation of this action if sight is to be saved.

These comments on conservation of vision and prevention of blindness serve only as an introduction to what may be a most crucial problem before us today—the strengthening and coordination of our various services. If, in our various professions, we are genuinely concerned

about helping people, there will be no need for professional gain. We should not have a desire for vain glory, we should not encourage professional demigods, we should not tolerate interprofessional jealousies or hostility, we should not create defensive reactions. We should learn to communicate with each other more meaningfully and to use each other more effectively; then we can discover new horizons for the prevention of blindness and conservation of vision.

With or without sight, we must have vision.

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CLINICAL ASPECTS OF NYSTAGMUS

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IN his book, Cogan² defines nystagmus as "a disorder of eye movements characterized by involuntary oscillations that are to a certain extent rhythmic." He then says, "This definition is not very precise but neither is the entity of nystagmus." To this might be added, "and neither is the treatment." I thought it might be possible to bring out some points which would be helpful to those of us who, as practitioners in ophthalmology, are expected, at least by the patient's parents, to do something about this condition.

Nystagmus is variously classified. From a clinical standpoint the most important is the etiologic classification consisting of (1) ocular, (2) vestibular, (3) central, and (4) congenital idiopathic types; and Cogan's more recent classification based on objective characteristics in which he has two main classes, pendular nystagmus and jerk nystagmus. Pendular nystagmus corresponds to the ocular type and jerk nystagmus consists of (1) optokinetic nystagmus, (2) vestibular nystagmus, (3) nystagmus from neuromuscular insufficiency, (4) congenital nystagmus, and (5) latent nystagmus.

When a patient with nystagmus is first seen, clinicians usually start thinking of possible etiologic factors just as they do in any disease. They consider possible specific causes such as central nervous system lesions, vestibular lesions and conditions of the eye causing poor

central vision. In searching for ocular causes the physician must look for abnormalities such as bilateral central chorioretinitis in infancy, albinism, congenital aniridia, congenital cataracts and corneal opacities. These abnormalities are fairly obvious on examination.

The central and vestibular types of nystagmus are less obvious etiologically, but attention to the exact nature of the nystagmus and neurologic and otologic consultation will usually classify the nystagmus properly. For example, rotary nystagmus is most frequent in the vestibular type and vertical nystagmus is almost always central in origin. Both of these are biphasic with the quick component always in the same direction. Pendular horizontal nystagmus is typical of the ocular type, which usually becomes biphasic on lateral gaze due to neuromuscular factors with the quick component in the direction of gaze.

Actually, many of the patients with nystagmus who are referred to us are simply patients with congenital nystagmus without any known cause. In most cases they have already been seen by an internist or pediatrician, specific extraocular lesions have been eliminated as possible etiologic factors and the patient is usually in good health. The nystagmus may be either pendular or biphasic and is usually horizontal, although there can be a rotary component. None of these patients have any evidence of eye disease present to account for the nystagmus. Often, a

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fairly high degree of hyperopic astigmatism is present but it is no higher than that of many patients without nystagmus and, usually, proper correction will give relatively good visual acuity. In fact, several authors say the visual acuity may be 20/20. In my experience, it has ranged from 20/30 to 20/50.

When a patient with congenital nystagmus is seen in the office or clinic the immediate reaction on the part of the examiner is all too often a feeling of futility. Most frequently, the tendency is to do a refraction and let it go at that. For example, in an issue of the *Journal of the American Medical Association*⁷ of several years ago advice in regard to the treatment of congenital nystagmus was asked of the editor. The answer was that, aside from careful refraction and correction of the refractive error, nothing is available for the treatment of congenital nystagmus. Certainly such a conclusion should not be drawn in any case until it has been analyzed thoroughly and some form of appropriate treatment attempted.

In examining the patient with congenital nystagmus, certain things should be noted:

1. Is the nystagmus of the pendular or jerk variety?
2. Is it horizontal, rotary or vertical?
3. Is it greater in one field of gaze than another?
4. Is the visual acuity best in a direction other than primary?
5. Is the head held in an abnormal position?
6. Is there a tropia?
7. Is the nystagmus increased on covering one eye?

The first two points have already been considered. The next three may be considered together. If the head is held in an abnormal position, the nys-

tagmus will be greater in one field of gaze than in the opposite. The position of rest, i.e., least nystagmus, is away from the position of frontal gaze, the eyes being turned toward the side of the lesser nystagmus. To compensate for this the head is turned in the opposite direction so that the position of the least nystagmus is central. The visual acuity is improved accordingly when the head is held in this position. It is important to consider this in checking the visual acuity. If the patient is made to hold his head straight, the visual acuity will be less than if it is held in the position of choice. For this reason it is best to use a trial frame rather than a refractor when doing a refraction.

Head turning may occur more often than is commonly recognized, but in varying degrees so as not to be too noticeable in many instances. When the turning is marked, the case probably then falls into the category which has been called congenital eccentric nystagmus. This will be discussed later.

The presence of a tropia should, of course, be looked for. Frequently an esotropia is present. If found, and the visual acuity is fairly equal, the patient should be put before the amblyoscope with the arms set at the angle of deviation. Electra Healy⁵ discussed four cases of congenital nystagmus with esotropia in which the nystagmus was stopped completely when the troposcope was set at the angle of deviation. The arms were locked at this angle and rocked from side to side. When ten degrees of lateroversion had been secured with the nystagmus still controlled, efforts were directed toward obtaining some degree of amplitude. Eventually, full excursion was attained from side to side and then all efforts were directed toward obtaining amplitude.

Such orthoptic treatment may reduce the angle of deviation and keep the

nystagmus under control. When an angle of deviation persists, surgical treatment of the muscle may be employed.

If either eye appears to be amblyopic, the physician should be sure the nystagmus does not increase on covering one eye. If this happens, it may be that it is really a modified form of latent nystagmus. At least the physician should attempt to disprove the presence of amblyopia by checking the corrected visual acuity with the head turned in the direction of the fast component. If an actual amblyopia is present, the physician should occlude the other eye and stimulate the macula with the head turned so that the nystagmus is at its minimum.

Typical latent nystagmus is not present when both eyes are open. On covering either eye, a nystagmus occurs with the fast component toward the side of the open eye. The increase of an already existing nystagmus by occlusion may be considered as a modified form. Its importance in testing the visual acuity of each eye is obvious. With the onset or increase of nystagmus, the visual acuity diminishes so that a false finding is obtained. However, if the condition is recognized as one of latent nystagmus, the proper visual acuity can be obtained by turning the head so that the open eye is adducted on looking at the chart. In this position the nystagmus is minimized and the visual acuity is markedly improved. This condition, its mechanism and probable causes are admirably discussed by Kestenbaum.⁶

In regard to congenital eccentric nystagmus, the position of rest is markedly to one side, causing considerable turning of the head. In a study of this condition Anderson¹ felt that the condition was due to injury to or lack of development of the cortical centers controlling conjugate deviation, thus upsetting the balanced tonus of all the

muscles working together. He elicited a history of difficult labor at the birth of these children and thought the condition was caused by birth injury or anoxemia of the newborn. Although this is a possible explanation, usually no other evidence of brain injury is present in these children.

Apparently there is a relatively greater tonus of the yoke muscles which rotate the eyes away from the primary position. In order to bring the head back to its normal position it is logical to weaken the muscles which have excessive tonicity. Recessions of the rectus muscles which are producing the slow movement in the position of greatest nystagmus must be done to accomplish this.

Anderson discussed several cases in which he used this procedure with good results. My own experience with this procedure is limited to one case, but the result is quite encouraging. A 5-year-old boy had a jerk nystagmus which increased on looking to the left. On gaze to the right, it was much less marked. At first (August 1955) it was difficult to get his visual acuity. As far as I could tell it was 20/50 for the right eye, and 20/100 for the left eye, with lenses as follows: right eye, $+50 +1.75 \times 80$; left eye, $+50 +2.00 \times 105$. Prism cover test showed 8 prism diopters esophoria for distance with and without correction; 8 prism diopters exophoria for near with and without correction. During 1956 it was noted that he tended to turn his head to the left. Corrected visual acuity in this position was 20/40 in each eye.

He was given orthoptic exercises during most of 1956. His ability to fuse improved and his nystagmus became much less marked on near vision. But the head turning became worse.

On looking through the literature I came across Anderson's article and was encouraged to try the same procedure.

On December 6, 1956, a 6 mm. recession was done on the right lateral rectus muscle and a 5 mm. recession on the left medial rectus. At the present time there is much less head turning. In fact, his mother states that she notices it only occasionally now. He fuses well for distance and near and has no nystagmus at all for near vision.

Finally, the psychologic approach in cases of congenital nystagmus is very important. Both parent and child should be reassured and encouraged. Many of these children do well in school. In fact their reading ability may be excellent, since in many cases the nystagmus lessens with convergence. Undue strain at home and school should be avoided. When head turning persists in spite of previously outlined treatment, parent and teacher should be

advised to permit the child to keep his head at the position of choice.

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SURGICAL TREATMENT OF THE DOMINANT EYE IN SELECTED CASES OF STRABISMUS

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I

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IDEALLY the ophthalmic surgeon should combine the talents of the orthoptist, the psychologist, a policeman and, perhaps least important, a surgical technician. When the day of the operation finally arrives, the patient at hand should be so completely analyzed that the procedure of "doing something to one or two eye muscles," as expressed by America's inspirational oculist, the late Dr. Richard G. Scobee, becomes a very simple procedure.

Unfortunately, we seldom seem to have the ideal situation. First, we find that all orthoptists are not in complete agreement on all phases of their art, that the child, the parents and the home situation pose a multitude of problems, and, lastly, that the ophthalmic surgeon does not possess all of the sterling qualities of infinite time and patience. Most important, he finds that his colleagues are not in complete agreement on how a specific condition should be surgically corrected. All of which points up the fact that the most successful team of orthoptist and oculist is the one which strikes the best compromise

with the existing conditions, giving the best results with the least amount of time and expense to the patient.

For a number of years we have been disturbed by the inconsistency observed in the results of treatment of the child with esotropia who has amblyopia in his squinting eye. The amblyopia is treated by proper glasses, if necessary, and occlusion of the dominant eye. Slowly the amblyopia recedes, and finally the orthoptist and the surgeon agree that surgical treatment of squint is necessary in order to obtain fusion and binocular vision. But on which eye will the surgeon operate? Almost without exception, most oculists today feel that given the above set of circumstances, the first operation should be done on the weaker, nonfixing eye. This is a rule of thumb, a dictum based presumably on the fear that one might hurt the good eye. If this procedure is carried out, what is the usual end result? The amblyopic eye is occluded at the time of the operation and is postoperatively occluded for days, or perhaps several weeks, not only because of the pain and discomfort of the operation, slight as they might be, but also because of the serious psychologic deterrent to the good vision which presumably had been built up by the preoperative occlusion of the dominant eye.

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How different this whole picture would be if the operation were performed on the dominant eye. In such cases, while surgically correcting the angle of squint we would, at the same time, be very effectively treating the amblyopia by occluding the dominant eye. By so doing, we would be giving the amblyopic eye a tremendous stimulus psychologically, and actually the stronger, fixing or dominant eye would not in any real way be threatened or exposed to any risk because of the operation.

Therefore, with this very common type of squint problem, a concomitant esotropia (seldom an exotropia) and amblyopia, serious thought should be given to performing the operation, not on the amblyopic eye, but on the fixing and dominant eye.

I began performing surgical procedures on the dominant eye before we had the high quality of orthoptic assistance that is now our good fortune. This surgical approach was adopted chiefly because it was the procedure that gave the best results most quickly. A little explanation was required to the parents in all cases, especially when the child came down from the operation with the bandages on the good eye. However, when it is explained to the parents that the strong eye was patched before the operation in order to strengthen the weaker eye, the logic of still keeping it patched for a while after the operation is clear to the parents, especially when the concomitant factor can be so easily demonstrated to them by the cover test before the operation.

Miss Sealey and I have a series of cases which are striking in the high percentage of postoperative satisfactory binocular vision with only one opera-

tion. This is in sharp contrast to the standard rule of thumb of surgical treatment of the weaker eye, which she has observed both in England and in America. As an orthoptic technician, she is particularly gratified by the consistent finding of sustained good vision in the amblyopic eye postoperatively.

CONCLUSIONS

For the orthoptist who has noted the poor convergence in postoperative bilateral recessions, or who has to start working on the amblyopic eye again postoperatively because the surgeon always operates on that eye, it may be the time to suggest to him a muscle shortening and lengthening procedure on the dominant eye.

For the surgeon who plans to correct squint surgically in several stages, exhausting the available muscles in the squinting eye first, this may be the time to take a second look at a logical approach to a one-stage procedure which may be most satisfactory to surgeon, patient and orthoptist.

Not all surgical treatment of squint should be done in the dominant eye. However, the purely mechanical approach, operation on the deviating eye to obtain cosmetic results, must be amended if surgeons are to improve their end results in this type of squint and obtain fusion, binocular vision and stereopsis.

II

MISS SEALEY

We have been working for only a short while with surgical treatment of the dominant eye to correct squint, so I have only three case histories that are at all complete. The other patients who have had an operation on the dominant

eye, have had it so recently that I do not feel that I can give a report on them.

Postoperatively these children are much easier to work with, since the single operation, in the majority of cases, results in the eyes being parallel or practically so. When the operation is performed on the weaker eye, two operations are often necessary to gain binocular vision.

CASE REPORTS

Case 1

Cathie M., 7 years old, was first seen in May 1956. She had right esotropia with slight right amblyopia and dense right suppression. She was wearing a small correction for hyperopia. Her left eye was occluded.

Preoperatively, Cathie had 25 prism diopters of esotropia with 8 prism diopters of hyperphoria in her right eye without glasses. She had six sessions of preoperative treatments and alternation. Normal retinal correspondence and some fusion amplitude were gained, but no diplopia. Occlusion of the left eye was continued until the day of the operation. A tuck and recession operation was performed on the dominant left eye.

Postoperatively, the patient had 15 prism diopters of esotropia and 3 prism diopters of hyperphoria in the right eye. Occlusion of the left eye was continued, since considerable suppression of the right eye remained. Fusion amplitudes were very poor. Later, in order to strengthen fusion, 8 diopter base out prisms were clipped on to her glasses. With these, she was able to fuse near and distance, although she occasionally developed right esotropia at near on accommodation. Cathie is still using the prisms while she continues orthoptic treatments in order to strengthen fusion.

Case 2

Shannon G., 7 years old, was first seen in December 1956. She had a medium hyperopia with a correction to normal, right esotropia with slight right amblyopia and dense right suppression. Preoperatively, Shannon's esotropia measured 40 prism diopters with glasses and 50 prism diopters without glasses. Occlusion was applied to the left eye and six preoperative orthoptic treatments were given. Before surgical treatment, Shannon had normal retinal correspondence, fair fusion amplitude and intermittent diplopia. Occlusion of the left eye was continued until the day of the operation, when

a tuck and recession were performed on the dominant left eye.

Postoperatively, Shannon had 12 prism diopters of esophoria with glasses and 22 to 30 prism diopters of esophoria without glasses.

Fusion amplitudes were poor. She usually had binocular vision, but occasionally the right eye still overconverged on accommodation. To alleviate this, the ophthalmologist gave her a full correction. I have seen her only once since then, and she had binocular vision at all distances with her glasses.

Case 3

Rickie J., 5 years old, was first seen June 1956. He wore a small correction for hyperopia. Rickie had left esotropia with left amblyopia, lack of normal correspondence and no fusion.

Preoperatively, his esotropia measured 35 prism diopters with glasses; without glasses he had 40 prism diopters of esotropia. No normal retinal correspondence, diplopia or fusion was gained in the six preoperative orthoptic treatments, and due to the lack of interest on the part of the child, we decided to go ahead with the operation. The right eye was occluded until the day of the operation, when a tuck and recession procedure was carried out on the dominant right eye.

Postoperatively, the patient had no esotropia with glasses and 4 prism diopters of esotropia without glasses.

The cover test at near showed slight left esotropia which persisted in spite of further orthoptic treatment. Again weak fusion was the problem. Clip-ons of 8 prism diopters were tried. The prisms produced fusion, so they were incorporated in Rickie's glasses in such a way that they could easily be reduced as required. He fuses for near and distance, but has exophoria for distance and esophoria for near. Fusion amplitudes are still poor, but convergence to a pencil is good. He now has worn the prisms three months and they are being reduced by 2 prism diopters, since he is now able to fuse with a total of 6 prism diopters. As his fusion strengthens, the prisms will be gradually reduced until there is no need for any prism at all.

All these patients had a common problem—weak fusion. But with time, growth, and further treatment we hope that their fusion powers will strengthen.

Another thing common to these patients is that postoperatively they have good and full muscle movements.

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THE ORTHOPTIST AND THE WORLD OF THE CHILD

DOROTHY SEYMOUR

PORTLAND, OREGON

EACH time an adult accepts and understands a child as he is at his own particular point of development, he is helping him along, in some measure, in his progress toward maturity. Orthoptists work with many children who have an added burden of growth to meet, that is, need for a conscious effort to achieve the binocular vision that most of us take for granted.

Dr. Grinker⁴ describes the eye as "a significant organ through which the developing child differentiates what is self and what is not self." As an organ assisting in early phases of development, both in separating the child from and uniting the child with the world around him, the eye takes on important biologic and psychologic significance. In our language, many idiomatic expressions illustrate our unconscious acceptance of the eyes' symbolic psychologic significance, as "your eyes are bigger than your stomach"; "she made eyes at him"; "give him the eye"; "he cast his eyes about the room"; "magic eye." People are all aware, quite consciously throughout their lifetime, of the feeling of anxiety that is associated with the thought of loss of contact and isolation from others through any serious impairment to vision.

Grinker also points out the importance of the eyes in maintaining contact with reality and preventing internal fantasy from becoming dominant. The

frequent serious emotional problems arising in people who, after eye operations, must remain blinded for some days or weeks illustrate this point. With the increasing knowledge of the stress and strain which separation from the parents, such as in hospitalization, puts upon a child, the orthoptist can recognize more clearly the added anxieties and loneliness that are heaped upon the child who has one or both eyes bandaged after surgical procedures. Unfortunately, too often, we expect him to adjust easily with quiet acceptance, rather than with the openly expressed feelings which are painful for adults to bear.

As orthoptists become acquainted with many children, they are particularly concerned that each patient be helped toward binocular vision in order to make the best possible use of his own pair of eyes. Miss Julia Lancaster⁶ suggests:

The child does not know that he is trying to get binocular vision; he does know that he wants to be comfortable, and he tries the easiest way he can find.

Dr. Irene Josselyn,⁵ well-known child psychiatrist, gives us a similar suggestion relating to emotional development:

While from the standpoint of society, behavior is either "good" or "bad," from the standpoint of the individual, it always has some positive value. The behavior of an individual represents the best solution for his conflicting drives that he has been able to formulate. This behavior is a manifestation of his attempt to adjust so that he may be as comfortable as possible.

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This concept of purposive behavior is sometimes pretty difficult to accept when an unruly child or irate parent makes life uncomfortable by complicating or resisting the help being offered.

But let's look at the child as he develops, both uniting with and differentiating from the world around him with these eyes of his.

From the ages of 2 to 6 the child struggles with unconscious conflict while relating himself to others. Freud designated this the oedipal period, with the child striving to become like the parent of his own sex and developing particularly strong feelings of love for the parent of the opposite sex. Here lies the early development toward the maturity of heterosexual interest which is necessary for sound marriage and mature parenthood. We see this quite clearly in outward behavior as the little girl cradles her doll and plays house, while the little boy turns to fire engines, airplanes and dump trucks. We cannot see here the profound workings of the unconscious sexual conflict which becomes the area of understanding of the psychoanalyst. But we do recognize that it is the child of mature parents who marches steadily forward through this period of psychosexual development, even as Christian in *Pilgrim's Progress* moved forward in his conflicts, not only with giants and dragons but with Mercy and Mr. Greatheart—on toward the Promised Land. Conversely, it is the child of parents still struggling with a sense of inadequacy at some or several areas of their own interpersonal relationships in whom we see fearful, aggressive, or otherwise inappropriate behavior. Thus it is that in family agencies and child guidance clinics, we have the opportunity to help in a three generation struggle. The mothers and fathers of growing children are seeking belatedly and admirably to understand

and work through the angers and fears and dependencies of their growing-up relationships, in order to be better spouses and parents now.

What shall we ordinarily expect of children and what of ourselves? The 4-year-old is considered in some instances to have sufficient attention span to make use of orthoptics; but I'd wager that in the world of the 4-year-old the orthoptist is a problem. I recall once hearing a child guidance clinic social worker grant that there were probably sound medical reasons for orthoptic treatments for a 4-year-old, but she wished there weren't sound reasons until the child was at least 5½ or 6. Why was this social worker so wishing?

Let's consider the 4-year-old. He has matured a lot since 3; he has discovered the world. Gesell² describes Four as "above all an age for going out of bounds." His body reacts muscularly as he listens; he likes to go from one thing to another; he does not like to repeat things, as he is busy learning new things about his physical and social world. This is a time of loquacity; he talks and talks about everything, questions persistently, and gives long, flowing responses himself. This is a busy mind and body that is demanding of and often taxing to the adults around.

The 5-year-old has developed more of a social sense along with greater motor maturity. The latter, Gesell² has found, is "reflected in the free adaptive manner in which he solves simple problems involving geometric and spatial relations." He is beginning to enjoy increased activity under stress of rivalry.

Six³ has a dramatic quality about it—with quickly changing moods and new domains of experience. The 6-year-old is learning to organize his feelings and thinking, as his body develops rapidly and his world expands socially. But he likes and needs rituals and stable con-

ventions in the midst of this expanding world—he'll begin to step or not to step on the cracks in the sidewalk. He will probably do a nice cooperative job with orthoptic exercises in your office, although he may be pretty quarrelsome at home if it is necessary for him to keep at them regularly outside of the office.

It's very important to understand pretty clearly what represents average, though often to the adult irritating, developmental behavior for a given age. Then we can recognize as part of growth the scattered attention span and loquacity of Four, the brashness and quick mood changes of Six, the self-absorption of Seven. (I didn't mention Five because he's apt to be so much easier than Four that he's hardly irritating at all.) Really understanding what a child has a right to be like also helps the technician recognize more clearly truly difficult responses, either in the child's adjustment in the office or in his relationship with the parent as she may observe it. Extreme behavior in either area, we know for sure, is an indication of more stress on the child from some direction than he can tolerate, and his acting out or withdrawn behavior is his way of coping with the stress. Has he recently started to school, with all its attendant social adjustment, as well as having to concentrate on eye troubles? Or does he live in a family that constantly provokes its members into eruptive behavior so that he has to find out where the technician's point of eruption may be? Or does he feel accepted as a person and behave beautifully with the technician, while his mother reports she can't do a thing with him at home?

The many combinations of problems in adult-child relationships cannot be discussed here, but there are some broad concepts that can be mentioned. First, the child has to adapt to his own situation, which in the technician's contact with the mother may look pretty good.

But the child has to be what the parent's unresolved conflicts will him to be. I recall a mother of excellent educational and economic background who brought her 3-year-old daughter to a child guidance clinic where I was then a staff member. She had been told the child needed help because, among other things, the mother could not leave her without a real scene taking place. At the clinic the mother came into the playroom with the child, but when asked to go to her own interview when the child quite quickly became absorbed in the play material, the mother attempted anxiously to draw the child's attention to her. During the next week, the mother called to say she did not plan to continue as suggested because, while she admired the competence of the caseworker seeing her child, she felt the child might become too attached to someone other than herself. We can see quite clearly that this mother's anxieties about herself are being imposed on the child. The strengths of a more secure mother will likewise be communicated to her child, who will thus be given courage and freedom to move into a broader world of people and experiences.

Second, the more any person likes and accepts people, each person as he is, the more readily a child, well or poorly adjusted, is able to like them and want to cooperate. Nevertheless, any human being, child or adult, tends to transfer to any helping person, be it in the medical, educational or social-work field, some irrational elements from other relationships. Except in psychotherapy, no attempt can be made to analyze or handle such transference of feeling. However, to recognize this as a possibility does much to lessen judgmental demands and to increase the real acceptance of others.

This brings us to a third important consideration—how well do we who

want to help other people know ourselves? Do we really have any idea why certain kinds of people get us so upset while we do pretty well with others perhaps more difficult? This response of ours to the people we work with is given the fancy name "counter-transference." Dr. Blank¹ of the New York Psychoanalytic Institute, in a paper given for professional workers in the field of prevention of blindness, had several things to say about this that I would like to share with you. He states:

Most of us in our training have to overcome counter-transference problems relating to excessive need for approval, a need to be the omnipotent, benevolent rescuer or to Play God. The worker with this type of problem unresolved will tend to prolong the client's dependence on him as long as the worker's unconscious need is gratified. He will become distressed and angry, and tend to terminate the relationship if the patient asserts himself and doesn't "behave." The more insecure the worker in his personal and professional status, the more he will tend to over-react to any implication of dissatisfaction, anger, or lack of progress in the patient. . . . Since no one is completely free of unconscious conflict, counter-transference will occur in every worker. The more mature the worker and the more insight he possesses into his own assets and liabilities, the less frequent and severe will be his counter-transference difficulties on the job as well as his interpersonal difficulties in general.

This, of course, is a more complicated way of saying that the better we know and accept ourselves, the more understanding and less judgmental we are of others.

This leads us back to the world of the child and his parents, and how we can help. Because of the psychologic and biologic significance of the eye, any impairment of the child's vision or

muscular imbalance is likely to produce some degree of anxiety or guilt in the parent who produced him. Furthermore, frequent visits to your office for the child, with perhaps the discipline of home exercises, too, may be almost more than an already busy mother can realistically handle. And the child who wears and has to keep track of glasses has one more thing he has to look out for that limits his strenuous activities; these things in addition to the tremendous job he already has of striving toward maturity.

So we can see that more and ever more deep understanding and respect for the integrated growth processes—physical and emotional and intellectual—of the human being can increase the usefulness of those of us whose job it is in a particular field to help with very special problems.

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EXAMINATION OF THE PRESCHOOL CHILD

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THE importance of the early detection and treatment of eye problems in children has been repeatedly emphasized. We are all too well aware of what happens to the child with heterotropia if an early diagnosis is not made and subsequent adequate treatment not administered. This is common knowledge to those engaged in ophthalmology and orthoptics, but I often have ample cause to wonder whether we are doing as much as we should in informing the remainder of the medical profession and the general public as to the importance of early care of the eyes of children. Most of the youngsters examined by ophthalmologists have been referred to them by pediatricians and general practitioners, who often are poorly informed as to how to recognize eye problems and of importance of treating eye problems in children as early as possible. Occasionally I am confronted by the distressed mother of a child with amblyopia due to suppression who states that she was told to wait until her child was of school age before taking him to an ophthalmologist, or to wait and see whether he would outgrow his crossed eyes.

At present, the various ophthalmic, optometric, and educational organizations are urging that all children of school age should be adequately screened for defects in vision. There is no unanimity of opinion between these

groups as to what method of examination or by whom the examination should be administered, and I do not intend to get into that controversy at this time. However, all three groups soundly agree that the school-age child should have an eye examination. I don't disagree with this suggestion, but why wait until the child is of school age? If we are ever to gain any headway in the prevention of such eye problems as suppression amblyopia and abnormal retinal correspondence, we must see and treat the preschool child and not the child of school age. By preschool age, I mean the ages from birth to five years.

Sometimes I feel that we also tend to forget the importance of early diagnosis and treatment of eye problems in the preschool child, and a brief review of the eye diseases common or more prevalent in the preschool age group is well worth a few minutes of our time. In the successful management of young patients with extraocular motor anomalies, the majority of which are present before the child is of school age, it is imperative that we see and treat these individuals before they develop abnormal visual reflex patterns, such as abnormal retinal correspondence and suppression amblyopia. Once these abnormal reflex patterns have developed, the length of treatment is extended and the ultimate prognosis worsened. Yet our eye screening tests are set up for the school-age child and not the preschool child; therefore

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TABLE I
HEREDITARY EYE DISEASES — MODE OF INHERITANCE

DOMINANT	RECESSIVE	SEX-LINKED	UNPREDICTABLE
concomitant strabismus	pure albinism	choroideremia	cerebromacular degeneration
Duane's syndrome	microphthalmia	red-green blindness	macular dystrophy
ptosis with epicanthus	amaurotic family idiocy	megalocornea	congenital absence of rods
phakomatoses	Laurence-Moon-Biedl syndrome	Leber's optic atrophy	congenital absence of cones
blue sclerotic syndrome	hydrophthalmos		keratoconus
cataracts	corneal dystrophy (macular type)		usually alternating with high astigmatism
retinoblastoma			craniofacial anomalies
glaucoma			hereditary ataxias
dacryocystitis			elastosis dystrophica
arachnodactyly			Groenblad-Strandberg syndrome
corneal dystrophy (granular and lattice type)			pseudoxanthoma elasticum
aniridia			nystagmus
recurrent corneal erosion			pigmentary degeneration of retina
heterochromia congenita			progressive external ophthalmoplegia
dominant optic atrophy of children			gyrate atrophy
			Fuchs's epithelial-endothelial dystrophy

several years usually elapse between onset of the child's heterophoria or heterotropia and the beginning of treatment. Whether the suppression amblyopia is a result of a heterotropia or a high anisometropia, I often get discouraging results in attempting to disrupt the amblyopia with ocular occlusion and orthoptic exercises if the child is not from the preschool age group, especially if the patient is over age seven.

Premature babies, in particular, should be closely watched by an ophthalmologist. They should have an eye examination as soon as practicable after birth; these babies are especially prone to the development of eye problems such as high myopia, heterotropia, glaucoma, cataract, persistent hyperplastic vitreous, and, of course, retrolental fibroplasia.

Early treatment of many of these disorders could mean the difference between normal or adequate vision and possible blindness.

Several types of tumors of the eye are particular to the preschool age child, such as the retinoblastoma, dermoid, dermolipoma, teratoma, angiomatous tumors, and the various phakomatoses.

Many, if not most, of the hereditary eye diseases are present at birth or become manifest shortly thereafter. The early detection of these diseases is important, not only because of the advantages of prompt treatment but also because we are then able to advise the parents of the mode of inheritance and of the likelihood of their future children having similar eye defects (table I).

As one can surmise from the preceding statements, the evidence is more than ample to warrant eye screening examination of preschool children, but why is it that all too frequently the child is of school age before he has had an eye examination? First, the importance of early eye care in preschool children is

not appreciated by the remainder of the medical profession and the general public. Second, ophthalmologists are somewhat reluctant to examine and give an adequate diagnosis in these young patients because such an examination is time consuming, occasionally requires the use of a general anesthetic, and frequently offers only inconclusive evidence as to the visual acuity of the child.

Because of the excellent rapport that frequently develops between the parent of a child receiving orthoptic exercises and the orthoptist, the parent may ask questions or reveal information that he or she would be reluctant to discuss with an ophthalmologist. For example, "Should I have the eyes of my other children examined?" "Is this condition hereditary?" and "A friend of mine has a child with an eye disorder; when should she have it treated?" A few minutes of wise counsel at this time may be of utmost importance to the future vision of the child.

A second, and a very important, way in which the orthoptist can aid in this problem is in the designing or devising of better instruments or methods to use in examining the preschool child. I have always been impressed when visiting an orthoptic technician's office by the inventive talent of the orthoptist in designing devices in order to get better cooperation from the younger patient. It would not be too great a step to improvise eye tests to examine the visual acuity of the newborn child, such as the Massachusetts vision test which is used on the school-age child. Advances have recently been made in that direction; Drs. Gorman, Cogan, and Gellis of Harvard Medical School have designed an apparatus consisting of alternating black and white strips of varying thickness, simulating the widths of figures on the Snellen chart or E chart, which are mounted on the inside of a cylinder that rotates around the infant. Visual

responses to the moving strips were correlated on sixty newborns; four failed to respond. Two of these were known to be blind.

SUMMARY

The eye screening tests presently employed are designed for the examination of the school-age child and not the preschool child.

Preschool eye screening examinations are strongly recommended for the following reasons:

1. The extraocular motor anomalies generally are present at birth or develop long before the child attends school, and several years may elapse if treatment is delayed until the child is of school age.
2. The child who was born prematurely is especially prone to eye disorders at or soon after birth.
3. Several types of tumors of the eye develop particularly in the preschool group of children.
4. The vast majority of hereditary eye diseases become manifest before the child is of school age.

Orthoptists can play an important role in preventive eye medicine if they will (1) encourage preschool eye screening examinations of children and (2) develop eye screening equipment applicable to the testing of the preschool child.

ORTHOPTICS: A CLINICAL VIEW

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ONCE in a while it is wise to look back at what we have done and to evaluate our results with an open but critical mind. For this reason, fifty consecutive patients, selected in alphabetical order, have been reviewed. All of them were referred to the orthoptic department for good reasons; all were private patients. The reasons for referral and the results of treatment will be discussed.

Many authors have presented their views on the selection of patients for orthoptic referral and treatment.

Cooper⁶ points out that, in treating patients with heterotropias, the patient must be both young enough to re-educate and old enough to have an adequate attention span. He also states that the child must have had some normal binocular experience before the onset of the squint. Similar views are expressed by J. R. Anderson¹ with emphasis on early surgical correction, but only when the patient is older than 2 years and when the deviation is constant, greater than 15 degrees, and not controlled by adequate refractive correction. He feels that a deviation is helpful in assessing the recovery of sight in amblyopic eyes during the course of occlusion.

The role of the orthoptist is clearly stated by Lorna Billingham.² She feels that the orthoptist should be able to diagnose the type of deviation (e.g. palsy or overaction), assess the grade of

binocular vision present, give a prognosis and treatment when necessary. She points out that considerably fewer cases of anomalous retinal correspondence occur when the patients are seen early by the orthoptist. This role is probably what most ophthalmologists expect of the orthoptist for, as Mary Ellen Burns⁵ says, "In my experience, patients are referred for 'Orthoptic Treatment' when the referring doctor really means, 'If this is an orthoptic case the patient is to have orthoptic training.'" I think the concept is entirely reasonable; it is frequently impossible to make a diagnosis without some treatment, a therapeutic trial as it were.

Little reference is made to the orthoptic treatment of adult patients. A note by Burian,³ however, points out that a number of adults with minor phorias and little disability can be made more comfortable with a few sessions of training.

In discussing the scope of orthoptic training Burian⁴ has summarized very nicely the "can do's and can't do's" of orthoptics.

Orthoptic training can:

1. Improve the elements necessary for normal monocular and binocular vision
2. Correct pathologic exaggeration or deficiency of these elements
3. Encourage proper visual habits

Orthoptic training can't:

1. Cure strabismus
2. Help a paralytic or mechanical squint

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3. Compel fusion in someone incapable of fusion

One other noteworthy statement is made by Stallworth.⁷ "If no progress is being made toward fusion after a reasonable trial period with good cooperation from the patient, this fact should be admitted readily and orthoptics discontinued." I would go further and stop treatment in face of poor cooperation.

The fifty patients were referred to the orthoptist for one of three reasons.

1. Preoperative or postoperative evaluation

2. Treatment to improve fusion, to eliminate suppression, or to eliminate anomalous retinal correspondence

3. Symptoms of convergence insufficiency or phoria

A tabulation of these referrals is very interesting.

A. Age Group:

1. children 5-10 yrs. 29 (58%)
2. teen-age 11-20 yrs. 4 (8%)
3. adult a. 21-30 yrs. 16 (32%)
b. over 30 yrs. 1 (2%)

As one would expect, the greater proportion are children, but it is surprising to note the number of adults in the twenties who required help. The children usually had heterotropia, and orthoptic training was considered as one phase of treatment. The adults were, to a great extent, people requiring symptomatic treatment for headache, asthenopia, diplopia and poor convergence. The most notable feature was the paucity of teenagers; apparently their squints have been corrected or ignored and their resilience is enough to overcome minor phorias without discomfort or loss of efficiency.

B. Type of Deviation:

1. convergent 28
2. divergent 22
3. accommodative 3
4. hypertropia 5
5. convergence insufficiency 4

The total exceeds fifty as all the hypertropias were either divergent or convergent, mention of hypertropia being made only when it was very pronounced. The convergence insufficiencies were, without exception, slightly divergent and were listed as divergent squints.

The small proportion of pure accommodative squints corresponds roughly with that found by other reporters, though no statistical significance can be inferred from such a tiny series.

It has always been a dictum that convergence is far more common than divergence; thus it is a little surprising to find such a high proportion of patients with divergent squint in the group. This may be explained in part by the fact that many convergent squints are obviously unsuitable for training, whereas almost all individuals with divergent squint have enough sense of fusion to allow treatment, if only for the purpose of reinforcing fusion and stereopsis.

C. Results of Treatment:

1. improved by orthoptic training alone 15
2. improved by orthoptic training and surgical procedures 16
3. unimproved 11
4. untreated 7
5. worse 1

Not reviewed: Those treated by surgical operation, occlusion or refraction without orthoptic training.

It may seem a little harsh to say that only 64 per cent of selected cases were improved. In this instance, improved means that the eyes were reasonably straight most of the time but, more important, had some usable degree of fusion. The percentage would be lower if only the patients with tropias were considered, without a number of adult phorias to raise the figure. The unim-

proved were those in whom no fusion was attained, though often vision of 20/40 or better was present in the afflicted eye. A number of the unimproved patients should more properly have been moved to the untreated group a little sooner than they were.

The one patient made worse by treatment is worthy of note. She is 23 years old and was treated very intensively as a youngster, both surgically and orthoptically, to the point of developing persistent but intermittent diplopia. I decided against any further treatment of any kind, since she has learned to cope with her diplopia as it currently exists and I feel that any shift in the position of the second image would merely upset her. This is an example of the triumph of enthusiasm over judgment.

DISCUSSION

In my opinion, all patients with squint require orthoptic examination, whether it be done by the surgeon with a simple cover test, flashlight and observation or by the surgeon's assistant, the orthoptist, with more expensive machinery. The more information available, the more likely is one to achieve a good result. Treatment, however, is not always advisable. Many patients can be dismissed after the first examination. For example, it is almost impossible to do more than occlude the eye of a child under 5 years old. For this reason few of the above patients were sent to the orthoptist until they were 5, and only then when vision in the amblyopic eye had improved to 20/40 or better. Patients with true alternation, those with intense suppression and those with fixed anomalous retinal correspondence do not benefit, and they comprise most of the untreated group and the unimproved group.

When to stop treatment, when to operate and when to continue treatment are questions which usually

answer themselves. Patients with mechanical squints need operation. Those patients who cease to progress but still retain a noticeable squint need operation. Those, who, despite improvement, have too great a deviation to control need operation. On the other hand, patients continuing to improve, with only minor deviations, should continue treatment to a point short of boredom and then, if necessary, take a break, to be followed by a later course of treatment. In this instance, the orthoptist must make the decision; it is beyond the power of the surgeon to evaluate the exact degree of progress of the patient with any certainty.

For patients with divergent squints, orthoptic treatment is very useful but has one defect: it gives the surgeon a false sense of the patient's ability to converge. The surgeon often gains the impression that convergence is excellent, whereas it is really only well trained. He may then do a simple recession of the external recti, when he should have done some shortening of the medial recti in addition. This defect may explain some of the secondary divergences following surgical operation.

Treatment of adults poses a different problem but often gives a very gratifying result. The necessity for treatment is usually indicated by symptoms. Almost all the adult patients have convergence insufficiencies or minor heterophorias, and almost all achieve striking relief within three to four visits, though they may require further aid every few years, particularly after illness. There is a danger in the indiscriminate use of orthoptic training as a placebo, while failing to go further in attempting to relieve a basic psychologic problem. Some mention of the use of orthoptic training in aiding presbyopic patients is noted, but I feel that effort spent in teaching the patient how to get along with bifocals is more rewarding.

SUMMARY

1. Fifty cases are reviewed and commented upon, without attempting a statistical analysis, since the series is far too small to be significant.

2. The proportion of patients with pure accommodative squints and convergence insufficiencies, who are unquestionably the best orthoptic patients, is surprisingly small.

3. Despite this small number, a high proportion of patients with tropias and patients with phorias were benefited by orthoptic training.

4. A degree of caution is urged in preoperative assessing of the patient with a divergent squint who has received orthoptic training.

5. Finally, I think it would be valuable to analyze and evaluate in detail the results of treatment in any large clinic, on a periodic basis.

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ANISEIKONIA

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ANISEIKONIA is a very controversial subject; many believe it does not warrant any special consideration.

Unfortunately, patients who may be suffering from aniseikonia are all too often typed as neurotics and usually travel from office to office seeking relief from their discomfort. If it is undetected, the patient must resign himself to a life with symptoms he must try to tolerate; when recognized, the answer is simple and the patient can look forward to reasonable eye comfort in the future.

The definition of aniseikonia is a difference in the size of images; but, interestingly, a definite size difference is not necessarily subjectively noticed by the patient. The present concept of aniseikonia, with which I definitely agree, is that it is a recessive, not an acquired, condition, always present but not always evident.

Since all visual processes are transposed in the optical centers of the brain, all adaptations to visual defects are also controlled. Many patients may have aniseikonia but are unaware of it because of the involuntary control exercised by the brain over this defect. Patients on a higher mental plane who have overworked themselves and therefore overtaxed their eyes, causing a breakdown in these optical centers, have made this condition manifest; the brain which formerly adapted to this condition is now unable to cope with the added strain.

The basic symptoms of aniseikonia are similar in character to those of convergence insufficiency. The symptoms are (1) complete physical exhaustion, (2) extreme photophobia, (3) definite intolerance to motion of any sort, which results in vertigo and possible nausea, and (4) severe headaches, or pain in the eyes, after use.

A patient with aniseikonia may be treated by the orthoptist and does seem to get temporary relief of his symptoms from the treatment. However, he eventually reaches the point where orthoptic training is of no benefit and, if continued, the treatment would only tend to increase his discomfort. In this way, orthoptic training is more useful as a process of elimination than as a cure in this condition.

Only after the patient's ametropia has been corrected by glasses or trial lenses in a trial frame, and all other possible contributing factors, such as physical disorders, emotional problems and muscle imbalances, have been eliminated, is a patient considered suitable for testing.

The problem of aniseikonia had been recognized for several years but it is only with the recent development of a suitable instrument that uncomplicated testing and correction with lenses has been possible.

The space eikonometer is the instrument used for testing for aniseikonia. It is designed to measure size differences in the vertical, horizontal, and oblique meridians by applying the principle of

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binocular localization in space of specially selected objects. The patient must appreciate some degree of stereopsis, no matter how small.

On the top of the instrument, at the front, are three scales and two control levers. The scale not associated with a lever shows the separation in millimeters of the two optical systems. The other two scales, equipped with magnifiers, show the horizontal and vertical magnification differences introduced into the instrument. Either magnification difference may be changed by the forward or backward movement of a lever.

The scale before the patient's right eye shows the horizontal (axis 90) magnification differences between the two eyes. The scale registers magnifications from left, 5 per cent axis 90 through zero to right, 5 per cent axis 90. Similarly, the scale before the patient's left eye shows the vertical (axis 180) magnification differences between the two eyes. It registers magnifications from right, 5 per cent axis 180 through zero to left, 5 per cent axis 180. Each small division of the scale represents 0.25 per cent magnification. On one side of the instrument is a declination unit control knob. Scale rotation of the knob changes the declination in the optical systems from zero degrees to plus or minus 1.7 degrees.

The eikonic targets before each eye are transparencies designed to give a three dimensional representation of five vertical lines and an oblique cross in space. Viewed through the projection lenses of the optical systems, while accommodation and convergence are adapted for distant vision, the patterns will fuse and appear stereoscopically in third dimension.

Optically, the red cross is projected about 10 feet from the patient. Two vertical green lines appear about 16 inches in front of the cross and two

vertical white lines appear about 16 inches behind the cross. The fifth, a vertical yellow line, passes through the center of the cross.

The amount of aniseikonia in a particular case is measured by introducing known magnifications by means of the adjustable magnification units and declination unit, until the target elements appear normal to the patient.

The testing must be done quickly to avoid tiring the patient and thereby affecting the validity of the readings. The patient should be instructed as to his role in the test. As it is a subjective test, there can be no misrepresentation on the part of the patient. He must be aware that the test involves binocular space perception and that the various elements of the target he sees in the instrument will seem to change their positions in space during different parts of the test. He will be asked to make careful judgments as to the relative distances and positions of elements. He is not to worry about his answers, but simply report what he sees.

When starting, the instrument is set at zero. The green lines and the cross are the only two elements of the design that the patient must concern himself with, and these are considered separately. As it is necessary that the axis 90 error be corrected before proceeding to the measurement of the axis 180 error, the patient must first observe the front vertical lines only. He is then asked, "Do the front green lines appear the same distance from you or is one nearer? If one is nearer, is it the right or the left?" If the right line appears nearer, the image in the right eye must be magnified and the lever of the axis 90 unit must be moved accordingly. Similarly, if the left line is nearer, then the image in the left eye must be magnified and the axis 90 unit lever must be moved accordingly. The limits of change in the magnification within

which the patient reports that the lines appear the same distance are then determined. In some patients this will be quite small, while others will be less sensitive. The midpoint range within which the patient reports that the lines are the same distance is the axis 90 horizontal correction.

With this axis 90 correction left in the instrument, the patient is instructed to observe the cross. "Does the cross, as a whole, appear rotated about the center line? If so, which side is nearer, the right or the left?" If the right side of the cross appears nearer, the image in the left eye must be magnified. Similarly, if the left side of the cross appears nearer, the image in the right eye must be magnified. By appropriate adjustment of the lever, the midpoint of the range of magnification, within which the patient reports both sides of the cross are equidistant, is determined. This is the axis 180 vertical correction.

With both the axis 90 and the axis 180 units left with the corrections found, the patient's attention is again directed to the cross. At this time, he is asked whether the cross appears inclined in space as compared with the center line (top nearer or farther away). The control knob must be rotated accordingly, until the cross is reported to be in the same plane with the center line.

The three measurements, axis 90, axis 180 and declination, should now be refined by repeating the above three steps in the same sequence, beginning with the three corrections found, rather than starting again with the instrument at zero. To break up any psychologic sets or tendencies, to suppress the clues to stereoscopic perception, the examiner should frequently interrupt fusion, for the moment, by turning out the light with the microswitch whenever changes are made in the adjustable magnification units.

As the patient's answers are bound to vary, I give a series of eight to ten of these tests and take the average of the readings as the final correction. The technician must remember that even this required aniseikonic correction is not in itself the measure of total aniseikonia, unless the patient requires no refractive lenses in the examination. The actual aniseikonic correction as designed in the finished spectacles is determined with relation to the refractive test lens used during the examination.

Case 1

A 26-year-old girl was employed in a responsible position in a bank. She was first examined in 1955, at which time she complained of severe headaches, complete exhaustion and a sensation of dryness of the eyes. She had very little refractive error, but an orthoptic examination showed a marked exophoria for near, remote near point of convergence, suppression of the left eye and poor vergence. Her doctor recommended that she start treatments for convergence insufficiency and gave her a prescription to relieve a slight degree of hypermetropia.

After three orthoptic treatments her symptoms increased. She became very sensitive to light, movement and concentrated focusing, and found it impossible to tolerate the exercises for more than fifteen minutes. Since convergence insufficiency seemed to be her chief complaint, she continued for a series of 21 treatments and, although she could still stand only 15 minutes on the instrument at a time, her headaches were relieved to some degree. But she had now developed severe pains in both eyes, particularly the left. She could tolerate a little more light but still had photophobia, and it was becoming increasingly impossible for her to carry on a full day's work. She was compelled to take a leave of absence for two months; during this time she received orthoptic treatments three times a week. However, even with complete rest and regular treatments, no marked improvement was evident. At this time it was decided to carry out tests on the eikonometer. Six tests were done, the readings were very consistent and the patient showed the need of a left over-all correction, axis 90 left 0.75 per cent, axis 180 left 1.0 per cent, declination 0.1 per cent, which was ordered. After wearing her aniseikonic correction for one week, the patient reported almost immediate relief of her most intolerable symptoms. The dull headache, with which she had been bothered for two years, disappeared and she was better able to adjust

to light and movement. She returned to work after two weeks. She is still under treatment for convergence insufficiency and is showing definite improvement, which would not have been possible without her aniseikonic correction.

Case 2

A 57-year-old woman was employed as a clerk in a department store. She was first seen in our office in 1952. At this time her refraction was -2.25 sph., -3.25 cyl., and axis 10 for the right eye and -2.00 sph., -0.50 cyl., and axis 125 for the left eye with $+2.25$ add for both eyes. She was very comfortable with this correction and easily adapted to the anisometropia. She returned for annual checkups of her refraction but had no complaints, and her error was found to vary only slightly. In 1955, she returned complaining of severe pains in the eyes and a feeling of nausea and exhaustion, and stated that her glasses felt as though they were drawing her eyes. She was in good physical condition and, although her refraction had increased slightly, the degree of increase was certainly not enough to cause the discomfort she was experiencing. An orthoptic examination showed a slight exophoria of two diopters for near. Her fusion angle on the instrument was at zero and her vergences and recovery points were excellent.

On questioning, she said that her position at the store had been changed. She was now employed as a cashier and was required to use her eyes considerably more than before. In addition to this more exacting position at the store, she had been spending her evenings reading to her husband, who was ill at home. She was given a series of nine tests on our eikonometer, which resulted in definite and consistent readings, and a final correction of left 1.50 per cent axis 90, right 2.00 per cent axis 180, declination 0.1 degrees was ordered for her. The patient received her glasses and was instructed to wear them constantly. She had a little trouble adjusting to them at first and was rather disappointed that they had not given her immediate relief. After wearing them for two weeks she reported that the pain in her eyes had disappeared. She was able to carry on a day's work in complete comfort and had come to feel such great relief with her lenses that she was afraid to be without them, even for a moment. She still reports to us regularly and is very comfortable with the lenses.

Case 3

A girl, 24 years old, was first seen in our office in 1944. She had intermittent exotropia in the left eye, with angle greater for distance than for near. She had had three years of orthoptic treatments for this condition before

coming to our office. Fusion and vergences were fair. She was found to be myopic without a cycloplegic and anisometropic with a cycloplegic. A recession of the left external rectus was performed. For the next five years, orthoptic treatment was continued and she showed marked improvement and was exercising good control over her eyes. She was given home exercises.

In 1952, she returned complaining of headaches and exhaustion after using her eyes and was finding it difficult to carry on with her grade twelve studies. The patient had fallen behind in her school work because of her eyes and had been attending night school in an effort to catch up. She was advised to stop school. Her muscle coordination was still good, and it was decided that eikonometer tests should be done. Eight tests were carried out and she showed definite readings. A correction of left 1.50 axis 90, left 2.00 axis 180, declination zero, was ordered. Her exhaustion was due to the conscious control she was exercising over her eyes; she found that with her aniseikonic correction she was much improved and able to relax this control. Her symptoms disappeared and over a period of time she was able to finish her studies.

We did not see her again until 1955. She had been taking an art course in Europe. She complained of pain in the eyes followed by nausea. These symptoms were relieved only by staying in a dark room and "sleeping it off." Headaches were becoming progressively worse and she suffered nervous exhaustion as a result of trying to see. Eikonometer tests were again done and there was a definite increase in her correction. New lenses were ordered as follows: left 2.75 axis 90, left 2.75 axis 180, declination zero. At the present time, the patient is employed at a part-time job and leading a normal life, free from symptoms.

Case 4

A woman, 37 years old, was first seen in our office in 1941. Her symptoms at this time were headaches, a drawing sensation in the eyes and drowsiness when she did close work. She was working the night shift in the telephone company, not as an operator but as a ledger clerk, and was required to use her eyes constantly for long periods at a time. She had no refractive error. An orthoptic examination disclosed an esophoria, greater for near than for distance, of three to seven diopters. She was given a course of orthoptic exercises and improved to orthophoria with good ductions, but was becoming exhausted from the effort required to control her eyes.

She was advised to stay home from work for six months. On re-examination, after six months of rest, accommodation and convergence had completely broken down and she was complaining of severe pain in the right eye after reading. Surgical procedures were performed on muscles to help relieve the patient's convergent spasm. The cosmetic result was good. The patient felt better after the operation. She continued with orthoptic treatments but the results were not gratifying, for although she sometimes showed marked improvement, there were also times when symptoms increased and she was forced to take periodic leaves of absence from work. From 1944 to 1948, many recognized methods of treatment were tried, such as bifocals, cycloplegic drugs and complete rest, but to no avail, and the patient's symptoms stubbornly persisted. At this time the first eikonometer tests were given.

The resultant readings were so small that it seemed impossible that this percentage of error could be the cause of her symptoms. However, she was suffering so much that lenses were ordered. There was some improvement in her condition with this correction but she still had a recurrence of symptoms when she did close work.

She moved to the country to avoid the stress of movement and glare in city surroundings. The patient had been advised to return for further examinations. Eikonometer tests were done again in 1952 and the left over-all

correction had increased. New lenses were ordered. She was now able to work, but her symptoms, though diminished to some degree, were still prevalent. She resumed orthoptic treatments and these proved more beneficial than before.

In 1956 she was again tested on the eikonometer and a marked increase in the correction was found. An over-all correction of left 0.75 axis 90, left 2.25 axis 180, declination $+0.1$ was ordered. This time definite immediate relief was appreciated. She is still receiving orthoptic treatment, and the esophoria which so stubbornly persisted has finally been eliminated. She is carrying on a full time job with little effort, but is continuing treatment in order to firmly establish good control of her vergences. We feel that this patient would never have improved to this degree without an aniseikonic correction.

The purpose of this paper has been to bring aniseikonia and its correction to your attention. It is my hope that in explaining the instrument and the method of testing and presenting several of our successful cases, I have sparked your interest in this subject so that you will consider its significance when endeavoring to eliminate all factors contributing to a patient's stubborn and persistent symptoms.

AMBLYOPIA EX ANOPSIA: APPROACH AND TREATMENT

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WE are all familiar with the look of horror and astonishment on a parent's face with the realization of Johnny's inability to see with his squinting eye. The parent has been quite conscious of his child's cross-eyed appearance for some time and yet never dreamed of a visual defect. Often such questions as "Will he eventually lose the sight in his other eye?" "What would cause the vision to drop?" and finally "Is there any chance of regaining the lost vision?" are all asked in rapid succession by the concerned parent.

Amblyopia ex anopsia is, as we know, a partial or complete loss of vision, purely functional in character, affecting the macular region only. It is the result of a suppression or exclusion from consciousness of one of the two images. In the case of strabismus the image of a fixated object falls on the macular area of one eye and upon an extramacular area of the other eye, or upon noncorresponding points. The resultant diplopia, as long as the strabismus remains, can be avoided only by suppression of the unwanted image from consciousness. Occlusion of the fixating eye in order to force the amblyopic eye to function is the means of treatment to regain the lost vision. Fortunately in most instances of childhood strabismus such treatment is successful.

When, during an eye examination, a child is found to have amblyopia ex anopsia and occlusion is prescribed, it should be borne in mind that occlusion of the good eye is a shocking and severe treatment. The young patient, who has been used to a 20/30 vision or better, through occlusion will be forced to work and play with as little as 20/200 vision. Such treatment is not without its psychological complexities and will undoubtedly be met by rebellion unless the child is treated in a very understanding manner.

First, before the occlusion is given, the defect must be explained to the child in terms that he may understand. Usually the explanation of a lazy eye is enough to give him some insight into the nature of his problem, namely that the eye is lazy and therefore weak and must be made to work to become as strong as the good eye. He should be told what to expect in the way of hazy, blurred vision when the eye is first occluded but he should be reassured that this will soon pass and that the sooner he has the patch over his good eye, the sooner his weak eye will be made strong.

The direct approach to occlusion is often the best one. The eye should be occluded before the child leaves the clinic, after the above explanations have been given. Often a light-hearted joke on the part of the orthoptist about making a pirate out of him helps to relieve the tension.

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Prolonging the anticipation of occlusion by waiting until the child gets home, or following the method whereby the duration of occlusion is lengthened each day—from two hours the first day to three hours the next day—are both poor techniques in that they only give the child longer to dread and rebel against such treatment.

Most children with sufficient understanding by parents adjust very well to occlusion. The parent should be informed as to his responsibilities toward the child. The occlusion will render the child handicapped for the time being, and more care and understanding will be needed in his supervision.

The technician should explain to the parent that as yet she is unable to determine the exact duration of the occlusion; that many factors such as age at onset, degree of amblyopia, duration of strabismus and present age are all factors influencing the length of time patching will be necessary. However, a more detailed and satisfactory prognosis could be given after seeing the child a few times and determining his rate of progress.

She should stress that traffic hazards, cruel comments by playmates and inability to do school work satisfactorily are just a few of the many difficulties the child will encounter. However, as far as possible the child should be treated in a very normal manner. He soon discovers the advantages his patched eye will render him in favors and excuses by oversympathetic parents and teachers.

If the child is of school age, often the teacher thinks she is being kind by excusing him from his school work when in actuality she is hindering his progress by denying him the use of his eye, the very therapy most needed.

The teacher should be urged to have the child sit in the front of the class and should encourage him to do as

much close work as possible.

At home the parents' responsibility requires that the child have added supervision, understanding and encouragement. It is the parents' responsibility to see that the constant occlusion is total at all times and that the child is encouraged to continue wearing the patch by showing him how much stronger his eye is becoming. The parent should encourage the child to use the eye as much as possible in performing such acts as reading, tracing, coloring and stringing beads, and to practice alternation.

When well-meaning but thoughtless friends and relatives inquire as to the wearing of the patch, the child should be encouraged to answer such inquiries himself in terms of "I have a lazy eye which is being made strong by wearing the patch."

The child with amblyopia should be brought into the clinic for vision checks about once every two weeks, and again the orthoptist should be generous in her praise and encouragement for any vision gained, regardless of how little. She is wise at such visits not to commit herself to the child as to the exact duration of occlusion, for the disappointment and discouragement of having to continue after promises of completion of occlusion should be avoided. Vague comments, such as "not too much longer," will suffice and yet avoid any commitment on the part of the orthoptist.

A short treatment to stimulate monocular vision is often given on the amblyoscope during clinic visits. The purpose is two-fold: (1) to stimulate interest on the part of the child to return again and see more pictures, and (2) to give actual stimulation to the amblyopic eye. However, treatments are usually not started in earnest until the vision is nearly equal in each eye; then intensive fusion training is given on clinic instruments.

Remember that occlusion should be continued until the child is capable of alternating. Regardless of equal vision, removal of occlusion without ability to alternate will only necessitate returning to occlusion at some later date. Once alternation has been mastered, the teaching of diplopia and fusion training are the next steps on the ladder leading toward surgical treatment, if it is re-

quired to correct strabismus.

In closing I want to stress the importance of early treatment in cases of amblyopia ex anopsia. The younger the child, the shorter is the duration, hence the recovery to normal vision is quicker.

After the age of 7 or 8 years improvement is much slower, becoming progressively more so toward adult life.

SOME PHYSIOLOGIC ASPECTS OF FIXATION DISPARITY

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THE study of fixation disparity involves the entire function of the binocular visual apparatus. Important subjects are:

1. The physiology of the macula
2. Representation of the macula in the cerebral optic cortex
3. The phenomenon of fusion with consideration of the horopter, Panum's fusion areas and stereopsis
4. The motor innervations resulting from the sensory stimulation and their variations in normal and abnormal alignments

Despite the fact that the phenomenon of fixation disparity has been demonstrated by many authors, its description in the standard orthoptics text is either brief or missing. However, it is often observed in normal individuals when they are tested on a haploscopic device such as the troposcope. Although the practical significance of fixation disparity in orthoptic treatment is probably limited, one should be familiar with its occurrence.

A review of monocular fixation will help define the limits of this process. The primary function of the extraocular muscles is to bring the object of regard upon the retinal area of sharpest visual acuity, the fovea. The eye generally continues to show three or four different types of movement even under the most exact conditions of fixation.

1. A fine vibratory movement occurring at the rate of 50 to 100 vibrations per second with an amplitude of 2 minutes of arc, which would be equivalent to anywhere from 4 to 6 cone diameters at the fovea.

2. Occurrence of waves 5 times per second through an amplitude of approximately 5 minutes of arc or 10 cone diameters at the fovea.

3. Rapid shifts or jerks from 5 to 12 minutes of arc occur once per second and would cover approximately 12 to 24 cone diameters at the macula. Adler states that the limits of fixation are probably around 20 minutes of arc at the macula.

4. Slow drifts.

The mechanism of fixation disparity is probably best demonstrated by a study of the horopter and Panum's fusion areas. For a given distance of bifoveal fixation it is possible to localize a frontal plane (horopter) from which all rays of light would fall on functionally corresponding areas within the two eyes. Objects lying on this horopter appear to be single because they are seen in the same direction by the two eyes. However, an object may be slightly in front of or behind the horopter and still not appear doubled. This binocular single vision is called Panum's fusional space. Objects lying closer than the horopter are viewed in temporally disparate directions and give

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rise to fused images which appear closer to the viewer. Fusion of nasally disparate images will cause them to appear beyond the fixation point. By this means, stereopsis or binocular three-dimensional vision is made possible.

Panum's fusional space also makes fusion possible without exact alignment of the visual axes at the point of fixation. This gives rise to fixation disparity which normally increases until diplopia occurs when the intersection of the visual axes exceeds the limits of Panum's space. The facultative temporary cooperation of functionally disparate retinal places may be called single vision within Panum's fusional area or panum vision. Prolonged observation and practice can narrow this area so that impression of objects imaged on such disparate places will appear more easily double. Panum vision is based on a flexibility of cooperation, a facultative unity of visual directions between an element in one eye and an element located within a certain area surrounding the anatomical partner in the other eye.

When the eyes are forced to converge, this convergence lags to the extent of the fixation disparity in the divergent direction. The opposite occurs in forced divergence.

The magnitude of this lag depends on the size of the peripheral fusion targets, training and cooperation of the subject and the nature of the individual's phoria.

The exact nature of this phenomenon is largely only theoretically understood. Improved physiologic research utilizing such methods as electroencephalography, electroretinography, electromyography, and other techniques, will surely throw more light on this subject in the future.

By one method, Adler's measurements of the physiologic fovea, in which the threshold discriminations seemed maximal, extended over an area of 250

microns (0.25 mm.) at the fovea, beyond which the acuity suddenly dropped. According to this conception, the acuity of vision does not increase to a point in the center of the fovea, but there is a plateau, which has measurable extent, throughout which the acuity is at a maximum. Other methods have determined much smaller foveal areas (30') to represent the finest points of visual discrimination; in all probability these measurements vary with the individual. However, if we consider the first datum, we see that the limits of fixation of a fine point might fall well within a fairly large area of the so-called physiologic fovea. On the basis of this reasoning, a fixation giving rise to high visual discrimination might take place in a choice of regions within an area up to almost one degree of arc before a slight blurring of this image would occur as it was fixed on the foveal margin.

The occipital cortex has a large portion of the striate area (area 17) designated to receive macular elements. Evidence obtained from lower primates has demonstrated that each linear unit represented in the macula is magnified 100 times when it reaches the striate area. When measured in the square, this would represent an area distribution 10,000 times that in the fovea. Therefore, if the limit of fixation of the fovea is roughly 20 minutes of arc (about 100 microns) and if this were magnified to an area 10,000 times this large, it would represent 1 sq. cm. in the striate area of the occipital cortex. It thus seems possible that a small object will be fixated in such a manner as to slip around in a 1 sq. cm. image projection on the cerebral cortex as the monocular fixation movements take place.

Therefore, both the retina and area 17 of the cerebral cortex may take part in the disparity behavior of certain visual impressions. Other areas which remain even less explored and which might contain a greater potential for

such phenomena are the peristriate areas, where the visual impulses are finally received and interpreted as visual impressions. Although some authors have felt that the striate area is the locus of the fusion center, associational fibers between the hemispheres are represented only in the higher or peristriate areas of the visual cortex and our final perceptual patterns are dependent upon the cooperative functions of these areas. To these last regions (areas 18 and 19) is assigned the task of giving visual direction to common retinal elements. The relationship between the retinal elements with common visual directions is fixed; for instance, after-images are always superimposed despite the effect of prisms, although the visual direction itself may vary with the so-called subjective metric of Tschermak. Tschermak feels that the localization of visual impressions does not occur with each eye, but that the impressions are jointly referred to our body as a tactile image and that to every singly seen visual object belongs a subjective direction of appearance, a common visual direction.

Elements of the visual organ are innately endowed with functional local signs, more specifically with functional values of sequence or order. Every retinal element has two definite functional values, a vertical and a horizontal value. These values are not constant, that is, the direction of subjective localization is not absolutely fixed. The local signs are merely values of sequence, members of a continuous gradation of order. Despite unchanging sequence values, the subjective metric can vary just as a pattern printed on a rubber sheet can be expanded or compressed even differentially in different meridians without disturbing the order of sequence.

Such a concept might help to explain the period of adaptation necessary for

individuals to become reoriented in space with changes in correction lenses. This is particularly noticeable in post-operative cataract patients who adapt to their new visual directions in varying periods of time. Fixation disparity might have components in levels of vision from reception to perception, i.e., retinal, striate, and peristriate regions.

The excellent work of Ogle, whose original investigations continue to throw much light on this and related subjects, has shown that the measurements of fixation disparity can be obtained to very high powers of discrimination. The relationships and practical aspects of treatment of small angle esotropia as compared to fixation disparity cases have been reviewed by Jampolsky. I refer you to these authors for review of the techniques used for measurement and determination of the finer amounts of fixation disparity and the findings in heterophorias.

This same phenomenon might also be applied on a broader basis, according to Dr. Swan. The term fusional disparity has been proposed as one which might be applied to that situation in which peripheral fusion may occur over a fairly large range of amplitude within which several degrees of fixation disparity might exist along with a suppression scotoma. The term fixation disparity, then, is probably best reserved for those cases in which normal correspondence is present without suppression. The limits of fusional disparity have yet to be defined from a measurement as well as a prognostic viewpoint.

As more is discovered concerning visual physiology from the point of image formation upon the retina to its perception and interpretation in the brain, with the resultant effect on the extraocular muscles, there will undoubtedly be revisions in older concepts and development of better understanding of the processes involved.

MOTOR AND SENSORY ASPECTS OF CONCOMITANT STRABISMUS

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THIS paper attempts to show the historic importance of Duane's explanation of the motor aspects of concomitant strabismus, which anticipated that of Breinin. It applies Breinin's theory to accommodative esotropia, to esotropia with convergence excess or divergence insufficiency, and to exotropia with convergence insufficiency. It deals briefly with the sensory aspects of strabismus. Duane's explanation of abnormal retinal correspondence is included as at least of historic interest.

The orthoptic technician is mainly interested in concomitant strabismus. In working with accommodative esotropia, innervational strabismus such as convergence or divergence excess, or with functional deviations, no orthoptic technician, I am sure, can resist asking, "Why is it that one eye remains fixing and the other deviates?"

The late Dr. Lancaster⁷ also asked: "What is it that determines the position of the deviating eye when its mate is fixing on an object? It seems to me," he continued, "that a clear answer to this question is very fundamental, and yet not generally clearly set forth by writers on strabismus."

A theoretical explanation of the mechanism by which strabismus occurs was recently expounded by Breinin.¹ He interpreted the innervational aspects of

convergence and divergence revealed by the electromyogram as showing that heterotropias are the result of a synthesis of vergence and version innervations.

An anticipation of Breinin's theory was expressed by Alexander Duane. Duane's explanation of the mechanism of strabismus is of interest in that he thought of strabismus as occurring through vergence and conjugate movements of the eyes.^{4,6} He says:

The essential element in this (i.e., esotropia) is a primary or secondary excess of convergence which makes both eyes tend to converge nearer than the point of fixation. Since rarely, if ever, is a convergence effort unequally distributed between the two eyes, both eyes in this case converge excessively and to the same extent. Hence instead of being directed at the point of fixation C (fig. 1), both are turned in equally to the nearer point A. . . but A will not appear clear since the eyes are not accommodated for it but for C, and the visual desire to get a clear image of C will compel one of the eyes to move in such a way as to get its image on the fovea. Suppose it is the left eye that does this. To do it, it will have to swing to the left until it points directly at C, and the image of the latter falls on the fovea *a*. This leftward swing is a conjugate movement shared equally by the right eye. Hence when the left eye has swung to the left through the angle ALC, the right eye has swung also to the left through an equal angle. . . , the left eye pointing toward C, the right deviating from the latter twice as far as before and pointing toward G.⁵

Duane's hypothesis seems to be correct in certain respects, but fails to state why these movements are not actually seen. No theoretical explanation is given of why the fixing eye makes no

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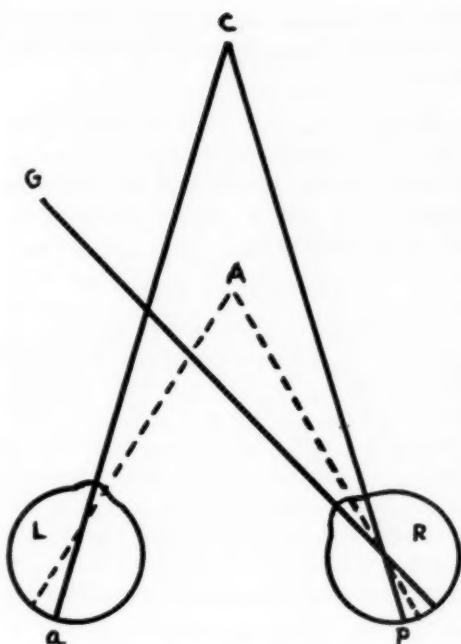


FIG. 1—(After Duane.) Convergence excess turns both eyes in to A; conjugate movement ALC, ARG swings fixing eye OS toward C, and OD toward G.

movement to A and then back again to C.

Breinin's theory, although formulated independently, is a simplification of Duane's. Based on his electromyographic findings of active excitation of the lateral rectus muscle in the deviating eye in exotropia, and following the clue supplied by Hering's explanation of asymmetrical convergence, it neatly answers the above objection to Duane.

Hering's law is applied to monocular motion as follows (from Maddox⁹) (fig. 2):

The lines y and z represent parallel visual axes, while the eyes are looking at some very distant object. Let the attention be now diverted to a near object c lying in the line of the right visual axis z, so that the right eye has no motion to make in looking at it, but the left eye has to sweep through the angle yAc. Hering has shown that half the motion of the left eye (γAo) is due to the converging innervation, and

the remaining half (αAc) to the innervation which turns both eyes to the right, and that while the two innervations conspire in the case of the left eye, they exactly counteract each other in that of the right.

Breinin¹ applied Hering's law to the active excitation of the lateral rectus muscle in exotropia, and said:

The fixating eye is in the primary position; the other eye deviates outward. The divergence impulse goes to the two lateral recti equally but is neutralized in the fixating eye by an opposite version. . . . Consequently, there is no observable change in the innervations of its muscles, whereas those of the deviating eye suffer the double dose of divergence and version.

It would appear that the same line of reasoning applies to the eso deviations.

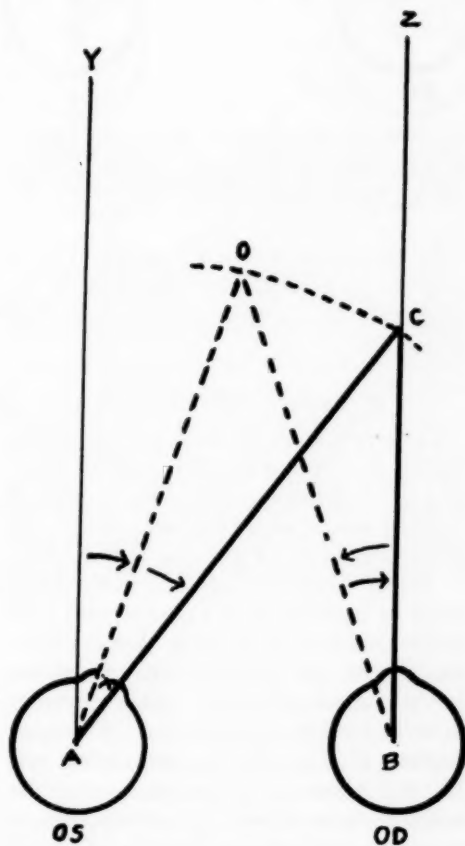


FIG. 2—(After Maddox.) Accumulative motion γAc of OS; dextroversion compensating convergence in OD.

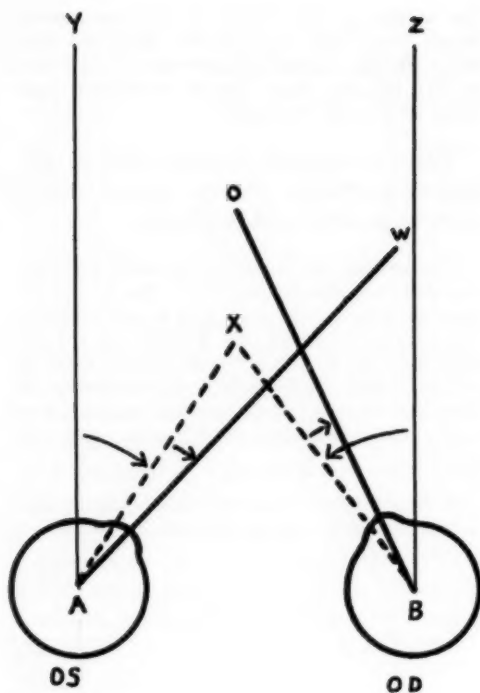


FIG. 3—Accommodative esotropia (or esotropia with convergence excess). Fixing eye OD receives convergence innervation zBX with dextroversion XBO to remain fixating at O. OS receives convergence innervation yAX plus dextroversion XAw .

In the light of the foregoing, it is of interest to analyze the situation in accommodative esotropia and other heterotropias.

ACCOMMODATIVE ESOTROPIA

Consider a child with an accommodative esotropia who has 3 prism diopters of hyperopia, and has a normal accommodation-convergence ratio. To fixate clearly an object one meter away, 4 prism diopters of accommodation must be exerted with a consequent convergence innervation of 4 meter angles. Since only one meter angle of convergence is needed for one meter distance, there is excessive convergence of 3 meter angles. If the right eye is fixating (fig. 3), the excessive convergence impulse must be neutralized by a dextroversion innervation to remain fixating. The left eye, on the other hand, will receive both

the convergence and dextroversion impulses and swing in to a manifest strabismus.

When correction is worn, the excessive accommodation-vergence reflex is brought under control and the eyes are straight, if other reflex factors are operating normally.

CONVERGENCE EXCESS

The above reasoning can be used to account for the position of the deviating eye in cases of esotropia with convergence excess. Here the excessive convergence is not due to a refractive error but to an excessive basic tonic vergence reflex. The fixating right eye (fig. 3) receives an excessive convergence innervation which is neutralized by a dextroversion innervation; the left eye re-

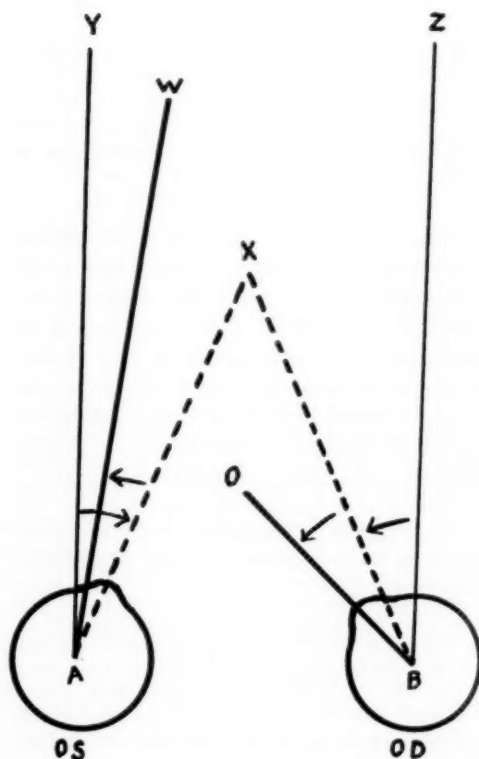


FIG. 4—Convergence insufficiency. Insufficient convergence innervation zBX , yAX brings eyes only to X. Levoversion XBO , XAw brings fixing eye OD to O, OS to w.

ceives the same innervations, now additive, and an esotropia is observed.

CONVERGENCE INSUFFICIENCY

In cases of convergence insufficiency, where the convergence reflexes are not sufficient to allow both eyes to fixate a given object O (fig. 4), the convergence innervation (zBx , yAx) would bring the eyes only to x . If the right eye is the fixating eye, a supplementary levoversion innervation (xBO) enables the right eye to fixate O. The left eye would receive the corresponding levoversion innervation (xAw) initiated by the right eye. This latter innervation would be synthesized with the insufficient convergence innervation and the left eye would deviate to w .

DIVERGENCE INSUFFICIENCY

When an esotropia has a greater deviation for a distant object than a near one, a divergence insufficiency exists. The following is based on a hypothetical case with orthophoria at near.

When transferring fixation from N at near to O at far (fig. 5), the insufficient divergence impulse NBX , NAX will bring the eyes only to point X. If the right eye is fixating, an added dextroversion innervation XBO is necessary to fixate O. The left eye will also receive the dextroversion innervation wAX initiated by the right eye. This innervation will be synthesized with the insufficient divergence impulse and the left eye deviates to w .

In cases of convergence or divergence excess, the impulses to the fixating eye are neutralized; in cases of convergence or divergence insufficiency, the impulses to the fixating eye are additive.

ORTHOPTIC APPLICATION

An understanding of Breinin's theory is an aid to the orthoptic technician when supporting the doctor's decision to perform binocular surgical procedures. Often parents balk at an operation on

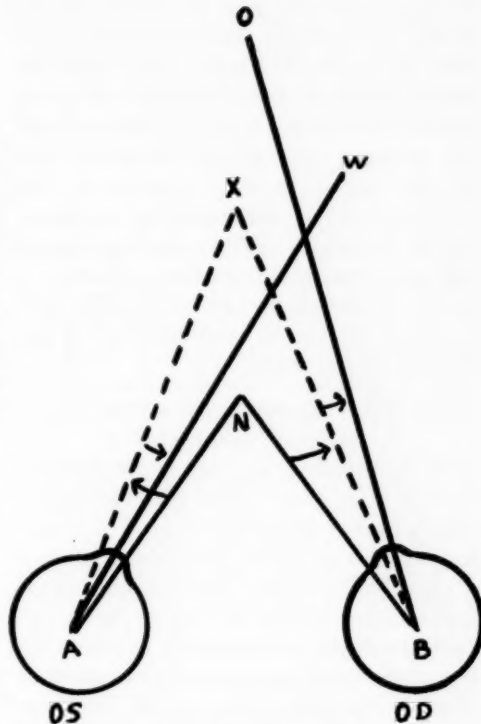


FIG. 5—Divergence insufficiency. Insufficient divergence NBX , NAX brings eyes only to X. Dextroversion XBO , wAX brings fixing eye OD to O, OS to w .

the so-called good eye. When the situation is explained in a simple fashion they become cooperative. Doctors have noticed that monocular surgery is likely to give incomitant results. Dr. Costenbader³ has said: "Patients having symmetrical surgery . . . have a higher degree of comitance following their surgery than do those who have asymmetrical surgery." An analysis of a hypothetical case of esotropia may be of value.

According to the law of reciprocal innervation, the innervations going to the fixing eye also reach its mate. In esotropia it is the excessive innervation going to the fixating eye which compels the other to deviate. In figure 3, if the right eye receives an innervation which allows it to fixate O, the same innervation will cause the other eye to deviate.

If surgical procedures are done on the deviating eye only, the innervations to the eyes are not altered. The right eye will still receive approximately the same innervation. If the left eye now becomes the fixating eye, displacement of the muscle might cause a change in the combination of innervations necessary for it to fixate. When the eyes need different combinations of innervations to fixate, incomitancy results. Fusion will be much harder to establish even if the discrepancy is slight.

RELATIONSHIP BETWEEN MOTOR AND SENSORY ANOMALIES

When dealing with concomitant strabismus, it is not always possible to decide whether a disturbance of the sensory system of single binocular vision or a disturbance of the motor system came first. Either can be the initiating factor. A disturbance of the motor system does not always lead to a manifest strabismus if all other things are equal; however when combined with a disturbance of the sensory system, such as blurred vision due to a refractive error, a manifest strabismus may occur. Similarly some people with a high amblyopia show very little muscle imbalance; one such patient had good stereoscopic vision when tested on the telebinocular stereoscopic target.

SENSORY DISTURBANCES IN STRABISMUS

Whether the strabismus is due to a disturbance of the motor or the sensory apparatus or both, a deviating eye will give rise to secondary sensory phenomena. These are diplopia, confusion, suppression and abnormal retinal correspondence.

Diplopia is due to the stimulation of disparate retinal elements by a single object; the object is then seen double. Confusion arises from the stimulation of the two foveas by dissimilar objects which because of the common visual direction of the two foveas are localized

at the same point. Suppression is the mental inhibition of disturbing sensory stimuli. In alternating strabismus the suppression is said to be facultative and the fixating eye acts as a conditioned stimulus for suppression in the deviating eye. In monocular strabismus the suppression is said to be obligatory since even when the deviating eye is made to fixate, it still suppresses; here again the fixating eye acts as a conditioned stimulus for suppression in the deviating eye.¹⁰

Abnormal retinal correspondence (ARC) is another sensory phenomenon of some strabismus cases. Usually associated with esotropia, it does occur with exotropia at times. Burian² has defined ARC as the condition in which "elements of the two retinas which are ordinarily disparate . . . prove to have a common visual direction." This definition is based on the theory that retinal elements have an innate spatial value and that each element has a subjective visual direction characteristic of it. Retinal elements in the two eyes which have a common visual direction are called corresponding points. Dr. Burian points out that the mechanism by which ARC develops has not yet been discovered.

Just as Duane's hypothesis of the motor anomaly in strabismus is interesting historically, so I feel his theory of ARC is interesting (although not necessarily correct). Duane has no special term for ARC, but calls unharmonious ARC "incongruous diplopia." His explanation rests on the premise that spatial localization is based on projection and muscle sensations. Utilizing his motor theory, he argued that if the deviating eye, say the right eye, escaped from the control of the convergence innervation and came under the control of the conjugate movements only, the eye would project monocularly. In that case the left eye (fig. 1) would project to C;

the fovea of the deviating eye would project monocularly to G. The image of C would fall on p. Monocularly then p would be projected to C. Because of the difference in the two sensory images of C, one clear and the other blurred, the latter would be actively suppressed. This, according to Duane, accounts for the deep suppression associated with ARC.⁵

This explanation is beautifully simple; but Tschermak and Burian have pointed out the difficulties involved in a theory of spatial localization based on projection of muscle sensations. Tschermak¹¹ does suggest that egocentric localization of objects may be the result of an "indirect sensory function of the ocular muscles."

In line with this, the thought occurs that the phenomena of ARC might be explained by the cortical association of egocentric localization of objects with the sensory aspects of asymmetric ocular-motor innervations. We agree with Linksz⁸ that the midline or straight-ahead direction is not an innate spatial characteristic of the foveas, but is determined by "the sagittal plane of bodily symmetry." An object will be perceived as being in the midline (if that is where it is) whatever type of ocular-motor innervations, but, in the case of an esotropia, would be associated with asymmetrical innervations instead of symmetrical. This would be true with either eye fixating. If the fixating left eye were to deviate, objects stimulating the left fovea would be localized to the right of the midline, because of the additional dextroversion innervation and because that is where they actually are. This

would naturally give rise to a conflict with stimuli arising from the retinal corresponding points of the right eye, i.e., the fovea. Cases of ARC occur when stimuli from corresponding points prove to be the weaker.

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THE USE OF MIOTICS IN STRABISMUS WITH EMPHASIS ON PILOCARPINE

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THE present treatment in strabismus therapy is the use of miotics. However, like everything else, we can go back in history to previous usage. In the 1890's Javal⁸ reports using pilocarpine and eserine so that some of his patients could leave their glasses at home when they went to a wedding. Vanity has changed very little in sixty years. This current trend was independently reactivated in 1949 by Abraham¹ when he reported using miotics in convergent strabismus. Since that time there have been numerous reports of miotic therapy here, in Europe, and in South America.

Probably the most important questions are how these drugs act and which forms of strabismus they help. Further, of the drugs that will produce miosis, which ones are successful and which is the best and safest. The miotics act on the ciliary muscle, as well as on the iris, stimulating accommodation. Consequently, accommodation is secured with little or no central accommodative innervation and a proportional lack of convergence innervation. In this manner, certain forms of convergent strabismus may be improved or completely cured. Obviously, miotics are useless in the treatment of divergent strabismus.

In order to understand the action of the various drugs we should delve slightly into their pharmacology. The

nervous impulse from the brain releases acetylcholine at the motor end plate so as to stimulate the action of the ciliary muscle. The acetylcholine is quickly neutralized by cholinesterase.

Physostigmine and neostigmine inhibit cholinesterase and do so in a reversible combination. Consequently, the normal acetylcholine can stimulate the motor end plate for a period of time without interruption. Both physostigmine and neostigmine are closely similar to acetylcholine chemically, which is the reason for the reversible combination with cholinesterase.

Next in line and similarly acting is DFP (diisopropyl fluorophosphate) an entirely different type of chemical compound. It forms an irreversible combination with cholinesterase. Therefore the acetylcholine can be active until more cholinesterase is brought from elsewhere in the body, or is manufactured locally. With this irreversible combination it is obvious why DFP is much more powerful and longer acting than any of the other miotics.

A third miotic is pilocarpine, which acts like the acetylcholine and stimulates the motor end plate until the muscle relaxes from the stimulation.

In summary, DFP irreversibly combines with and knocks out the cholinesterase giving a long powerful action. Physostigmine and neostigmine form reversible combinations giving a weaker shorter action; pilocarpine stimulates the muscle directly.

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The varied side reactions of the drugs are probably the most controversial and least understood of all the considerations. All drugs are expected to exhibit local and systemic allergic reactions. Knapp,¹⁰ using DFP, reported three patients out of 316, or 1 per cent, with allergic reactions. Two had local reactions and the third had an apparently allergic cough. Abraham³ also reports three allergic reactions in well over three hundred cases. One was a local reaction. The other two were systemic nervous reactions of bed-wetting, nausea, and yawning in one patient, and a nervous tic in the other. Both of these cleared when the drug was stopped. Malbran and Norbis¹¹ reported one case out of 109 in which DFP had to be stopped because of nausea and vomiting. In our series of thirty patients treated with pilocarpine, treatment was stopped in two instances; one patient developed hives, another had severe headaches. We have had a few patients on DFP, and one of them complained of such severe pain from the drops that she was hospitalized. However, in all fairness to the drug, it must be mentioned that she was a very neurotic girl. Consequently, it can be seen that any of the miotics will give allergic reactions in a small number of cases.

The other important side reaction of the miotics in general (and all of them will cause it) is the formation of iris cysts. Vogt¹⁶ first reported them in 1920 when he found glaucoma patients developing cysts after treatments with both pilocarpine and physostigmine. These cysts are brown or chocolate-colored bodies which appear, for some unknown reason, along the pupillary edge of the iris, usually on the nasal side. Probably the best clinical study of this problem was carried out by Abraham,⁴ who first brought them to our attention. In the 66 patients which he studied very closely for the cysts, 42

showed changes. The cysts were noted after one to forty weeks, averaging ten weeks. Usually, whenever cysts were noted, the miosis was very strong, whereas, an active pupil was noted in those patients who did not develop cysts. After cessation of the drops, the cysts disappeared in from two to forty-two weeks, with almost all cleared in ten weeks. Only small, slender tags remained. He did not find any cysts in patients treated with pilocarpine and only a few in those treated with physostigmine or neostigmine. Malbran and Norbis¹¹ reported that 75 per cent of their patients with squint developed cysts while taking miotics, which is markedly different from the 4 per cent that Gerewitz⁶ found on his glaucoma patients. Therefore, eyes of the young must respond differently from those of adults. Knapp¹⁰ also found the cysts in eyes with nonmobile pupils. He feels that in his regimen, which I will discuss later, no patient has developed iris nodules of significant proportions and only a few have developed nodules of even small proportions.

However, before we can feel that this is satisfactory, we must know more about the nodules themselves. What are they and how are they caused? I think that we must frankly say that we don't know. Originally the cysts were thought to result from the immobilization of the iris against the surface of the lens after a definite and prolonged miosis. This caused serous fluids to separate the two layers of the pigment epithelium on the posterior surface of the iris forming the cysts. This was questioned when Christensen, Swan and Huggins⁵ studied two eyes with these cysts, one pathologically. They had one patient with congenital aniridia and secondary glaucoma. Under miotic therapy he developed two typical pigmented nodules at the border of the rudimentary iris. As the iris was

immobile, miosis could not have been a factor in this case. They also studied the eye of a nine-year-old boy with congenital glaucoma whose eye was enucleated after the cysts developed because a tumor was feared. In this case there was a proliferation of the pigment epithelium with the formation of generally solid nodules rather than cysts. Further, the cysts were filled with a pigment-permeated fluid and not the clear serous fluid previously supposed. Also, a new growth of the iris pigment layer extended beyond the normal borders, an actual hyperplasia possibly bordering on a neoplasia. Obviously, much is still to be learned about these cysts and their long term course.

From the above facts on the pharmacology, we can see that drugs are primarily effective when the accommodation-convergence ratio is upset, with a consequent esophoria or esotropia. They have also been used as a diagnostic tool to save the expense of a trial on glasses.

Abraham² used miotics for treatment and reported, in 1952, on 132 patients with convergent strabismus. In 109 patients with intermittent or periodic strabismus, 87 per cent were helped. In the 23 patients with constant strabismus, 11 or 48 per cent were improved. Malbran and Norbis¹¹ reported similar results. They found improvement in 71 per cent of the patients with accommodative esotropia, 81 per cent of the patients with partially accommodative strabismus, and only 40 per cent in the patients with the nonaccommodative type. Knapp¹⁰ compared DFP and the use of glasses as forms of treatment in 123 cases. He felt that DFP was a better form of treatment than glasses in 10 per cent of the cases and equal to glasses in 78 per cent of the cases. Knapp also used the drug as a diagnostic aid in 126 cases. In these, he desired to eliminate the accommodative

element of the esotropia before deciding upon further therapy. Jonkers⁹ in Europe has also used the drug in a similar manner and feels that miotics are of very great diagnostic importance while their therapeutic effect is not too great.

Here at the Infirmary we have treated 36 patients with pilocarpine. Six patients have not been on pilocarpine for a sufficient time and three have discontinued treatment. Of the remaining 27, nine improved on the pilocarpine and 18 showed no improvement. Five of these 18 stopped the drops for various reasons; headaches, hives, and parental indifference. Of the nine which did improve, all used 2 or 4 per cent pilocarpine at least three times a day for two or more weeks. Two of the patients who were listed as no better actually improved some, but improved more with their glasses. We feel that good results can be obtained with pilocarpine, if the patient will continue frequent use of the drops. However, in some cases where pilocarpine doesn't quite do the job, Abraham² has suggested switching to DFP. In his series of 132 cases, ten patients who weren't helped with pilocarpine improved when given DFP.

In view of the above, what does the best regimen seem to be? Knapp has suggested using the DFP drops in each eye nightly for two weeks as a diagnostic aid. After two weeks the patient is re-examined. If there has been little or no reduction in the deviations, the accommodative component is regarded as minimal. If, however, there has been an improvement in, or elimination of, the deviation, the miotic is used every other night for two months. In the cases where this frequency has not succeeded, miotics have been stopped and glasses instituted. According to Knapp, this regimen has not produced any nodules of significant proportions.

Since the danger of nodules is still present, it seems that a slight modification of the regimen is indicated, considering the successes that Abraham and I have had with pilocarpine. After the initial diagnostic trial with DFP for two weeks, the patient could be treated with pilocarpine four times a day for two months. If this is not successful, the physician could always return to the use of DFP every other night, or even every night, temporarily. He must always realize that he is taking a small chance that the few small nodules might become dangerous at some future date. If the physician did not want to use DFP, the patient could be treated with glasses.

Miotics have proved beneficial in treating accommodative convergent strabismus, both diagnostically and therapeutically. Probably the diagnostic use will prove more important, in the long run, than the therapeutic use, especially since these same patients can be successfully treated by full cycloplegic correction, by bifocals, or by orthoptic training. Probably the miotics will be most useful therapeutically for temporary cosmetic improvement to permit the patient to remove his glasses for a limited period of time.

All the miotics have a place in strabismus diagnosis and treatment. DFP is by far the most powerful and consequently is much better for diagnostic use. However, this great strength of DFP, and the consequent side effect of iris cysts, make it desirable to use other drugs wherever possible. The occurrence of the iris cysts and pigment epithelium metaplasia must be studied further before they can be dismissed as insignificant tags.

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THE USE AND ABUSE OF PRISMS

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NEW YORK, NEW YORK

IN order to understand the use of prisms from the standpoint of refractive and motor anomalies, the purposes for which the prisms are used must be clearly understood. For this reason I have divided the phoria problems into two groups: (1) anatomic-physiologic and (2) induced.

The major causes of the anatomic-physiologic type of phorias are irregularities of insertions, discrepancies in the anatomic makeup, abnormalities of the check ligaments, neurogenic factors, and disturbances in the accommodation-convergence relationship. These phorias may also be due to temporary or semi-permanent systemic disturbances such as general debilitating diseases, anemia, endocrine imbalances, or pregnancy. In any of these situations a temporary phoria may be present which may become a permanent defect of the individual. As well as recognizing that the phoria is the result of an anatomic-physiologic problem, the physician must also know the degree to which the individual may overcome this particular defect. This is usually analyzed by interpreting the measurements on the screen and parallax test, the measurements with the Maddox rod, and the vergence measurements.

Some of the induced phorias are not necessarily induced by the refractive system, but may have an anatomic basis. They may be due to facial disparity:

asymmetry of the face, a high or a low bridge of the nose, protuberant frontal processes, or a head tilt. Many of these factors, combined with the use of glasses, may induce a phoria which could have been avoided had the facial disparity been considered.

In regard to the glasses or lenses themselves, the possibility of incorrect interpupillary distance or the misplacement of the glasses in front of the eyes must be considered for both distance vision and near vision. Occasionally lenses are ground too high up on the glass or placed too high in the frame. Bending the frames results in distortions, increases or decreases the proper astigmatic correction, and also induces prismatic effects. Tilting the frame produces distortions and incorrect decentration of the lenses.

Frequently, the method of using the glass will either produce or eliminate the possibility of induced phorias. If the person knows how to use a pair of lenses, he may be able to adjust to a situation in which he could not if the lenses were used incorrectly. In high degrees of anisometropia, if a patient looks through the center of the glasses when he looks down, he will not produce a phoria by the prismatic effect of the different lenses. However, if he looks down through the bottom of the lens, then a definite phoria will be induced. Bifocal segments are frequently offenders, particularly with the jump from the upper portion of the lens to the bifocal segment. In cases in which

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there is a large difference due to anisometropia, slabbing off is often used to prevent the induced phoria.

One of the more unusual but more interesting problems in induced phorias is the incorrect axis for the correction of an astigmatism. The eye will attempt to adjust itself to the improper axis of astigmatism by adjusting itself to the lens. In so doing the eye will have to be intorted or extorted according to the astigmatic axis. After a period of time the effect on the torsional element will also produce a vertical anomaly, and this anomaly can easily be measured. This problem is solved by replacing the astigmatic correction at the proper position; after a short period of time the induced phoria will disappear. One of the major problems in the production of the induced phoria is the lack of understanding between the ophthalmologist, the orthoptic technician and the optician. If serious problems were considered by this group as a team, a great many induced phorias could be eliminated.

The first step in treating phorias is to eliminate all induced factors. Study and analyze any asymmetry that is present. Evaluate head tilt to determine whether or not the head tilt is due to ocular causes. If it is, then the motility system must be treated; if the head tilt is the result of an orthopedic problem, it should be treated from that standpoint. If a head tilt is not considered when glasses are fitted, the inequality of the lenses to the position of the head will cause more problems. It is very important to decide on the proper interpupillary distance to be used. The interpupillary distance obviously differs for distance and near; the amount of difference must be determined and an amount somewhere between may be used in centering lenses that are to be used for both distance and

near. A pair of glasses for reading only will be decentered according to the near position, while lenses used only for distance will be decentered according to the distance position. The patient must be told how his lenses should be used in order to eliminate possible problems.

Second, the physician must always consider the strength of the lens as related to an existent phoria. A phoria should be measured with and without correction. Vergences should be taken with and without correction. Convex lenses reduce accommodative convergence stimulation, and concave lenses stimulate accommodation-convergence relationships. Decentration frequently may be used to reduce a phoria by slabbing off in the anisometropic lens.

Methods exist for using prisms in situations where they will be beneficial. Occasionally the prismatic correction is prescribed in relation to the particular muscle affected; whether the deviation is only for distance or only for near, and only in one eye or divided between the two eyes. This is the older method and perhaps the more frequently used. In considering the use of prisms, however, remember that no matter how large the prismatic difference may be or how large the phoria is, if the person is capable of overcoming the phoria, then small degrees of prism should be given at first and these increased gradually as necessary to relieve symptoms. Frequently, more than one system should be used in prescribing prisms, but a slow method is always preferable.

Prisms can be detrimental; they increase distortions, particularly in the periphery of the lens, and thereby cause additional symptoms in an individual. Moreover, although a prism will tend to direct the eye in a better position for binocular use, it is also a constant stimulus to the overaction that originally caused the phoria. Prisms physically correct the defect, but they also stimu-

late the process which originally caused it.

It is very difficult for a patient to wear prismatic glass if he does not wear his glasses at all times. The readjustment from prismatic lenses to no lenses at all is much more difficult than that from an ordinary pair of glasses to no lenses. When an increasing load of prism is expected, the patient should start with hook-over lenses as a temporary expedient until the proper adjustment has been achieved, one which rectifies the muscular disability and also totally relieves symptoms. These prisms may be given singly or in pairs. After a trial period, the effectiveness of the prisms should be analyzed on the basis of relief of symptoms, the tolerance of the distortion effect produced by the prism, ability to wear the lenses part time or full time, and the effectiveness of the prismatic adjustment for both distance and near. Perhaps one of the most practical uses of the prism is its use in preparation for surgical operation on the muscles. If a patient has a large

vertical phoria, he may gradually adjust to his defect with increasingly strong prisms. The prisms are changed until the prism correction can go no higher (perhaps 10 to 12 prism diopters correction over each eye). Then, if an operation is performed, the serious complications of the postoperative adjustment can be avoided. When the operation is performed without the preparation with prismatic adjustment, the patient must make the sudden adjustment on his own, and it sometimes is quite disturbing postoperatively. This method of using the prism is most beneficial to the patient, to the surgeon, and to the orthoptic technician. The use of a prism may also prevent a phoria from becoming a tropia.

Prisms should not be used (1) if refractive correction can handle the problem, (2) if decentration or a slab off will settle the situation, (3) if orthoptic stimulation can give satisfactory results, or (4) if re-establishment of fusion or relief of symptoms cannot be gained.

METHOD OF TESTING VISUAL ACUITY IN THE AMBLYOPIC EYE

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SINCE vision testing is time-consuming for the examiner and fatiguing to the child, any method directed toward more rapid and accurate testing of visual acuity is valuable.

The earliest age at which vision can be reliably tested is between 3 and 5 years. I have found the illiterate E chart the best method of testing the vision of a child unfamiliar with letters or numbers. Louise Sloan has shown that the Landolt ring corresponds in difficulty closely with selected letters.

When dealing with children, a small inaccuracy should be allowed for the sake of simplicity. It makes little difference whether the child points in the direction of the legs on the E or holds an E-shaped form, such as an E cut from cardboard, in positions corresponding to the ones shown on the vision chart. To make vision testing appear as a game is always desirable with children. Tell the child, "Now we are playing the E game." The procedure I employ with the young and apprehensive child is to encourage him to show me by pointing in the direction of the legs on the E keeping both eyes open. This simulates a game and avoids the apprehension of testing with one eye covered.

The examiner soon learns if the child's responses are reliable. As soon as the patient indicates the position of a

few large E's correctly and has gained confidence, the examiner occludes the left eye while the right eye is being tested. I prefer the projector chart because the test can be conducted with maximum facility and speed. The projector is adjusted so that only one letter can be seen at one time. Just as long as the letter is recognized without hesitation or errors I keep rotating to the next smaller line, showing only one letter on each line. The 20/30 line on the E chart seems to represent the usual maximum vision on the projector chart for children under 7 years old. The child is requested to indicate the direction of all the E's on the smallest line that he can read. One eye is occluded by the examiner while the vision of the other eye is tested.

A large proportion of children manifesting muscle imbalances are also amblyopic in one eye. An accurate measure of visual acuity should always be recorded. This is particularly important for those patients who are to receive treatment for the amblyopic eye. In many offices and clinics only one or two different charts are employed; these are usually charts similar to those used in most other clinics and offices, and consequently the 20/200 letter is soon known to be E or the numeral 3. Many clinic records indicate the vision to be 20/200 or 20/200-, which is grossly inaccurate. At present, visual acuity is seldom accurately measured when it is

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20/200 or less. One or even two letters are not sufficient for an accurate visual acuity test, especially when the defective vision of the eye is frequently tested, e.g., if the patient is under treatment for amblyopia ex anopsia. Only three visual acuity charts are available with letters ranging in size between 20/200 and 20/100. A method is needed for measuring the visual acuity of the amblyopic eye when the vision falls within this wide range; more important, a method is needed to evaluate the progress of the amblyopic eye under treatment.

The disadvantages of present charts and the standard method of testing vision at 20 feet can be remedied by using available charts at 10 feet or even 5 feet when the vision is subnormal. The testing distance of 5 feet is useful for the more severe visual defect.

The procedure for testing vision at 10 feet is either to bring the chart up to 10 feet from the patient, as in the case of a portable chart, or to walk the patient up to the screen when the projector chart is employed. For example, assume that the patient reads the 20/100 line at 10 feet, then the vision is recorded as 10/100. This is easily converted into the conventional visual designation of 20/200 by multiplying both the numerator and denominator by 2. Now, let us assume that the examiner tests the vision 5 feet from the chart and the patient reads the 20/50 line at 5 feet. The vision should be recorded as 5/50, because the patient is reading the 20/50 line at 5 feet. The visual acuity of 5/50 is readily converted into 20/200 by multiplying both the numerator and denominator by 4. The advantages of testing vision at 10 feet or 5 feet are:

1. No new equipment is required.
2. More letters to a line, therefore the test is more accurate.

3. Patient is less likely to memorize letters.
4. Method is flexible and test can be changed by using different distances and different charts.
5. Vision recorded on patient's record as taken at distances less than 20 feet indicates that more than usual care was exercised to obtain measurement of the patient's visual acuity.
6. Conversion to conventional designation is simple by multiplying the numerator or denominator either by two or by four, depending upon the testing distance of 10 feet or 5 feet.

The following proportion is a convenient method for recording vision in terms of 20 feet when any testing distance or test line is employed:

$$\frac{20}{X} = \frac{\text{distance at which vision is tested}}{\text{distance at which letter should be read in order to subtend an over-all visual angle of 5 minutes (standard vision)}}$$

(Example)

$$\begin{array}{r} 20 \quad 5 \\ X = 40 \\ 5X = 800 \\ X = 160 \end{array}$$

Therefore, 5/40 vision, that is the 20/40 line read at 5 feet, is equivalent to 20/160 visual acuity.

Vision as presently tested and recorded as 20/200 or 20/200- is frequently grossly inaccurate and carelessly taken. Defective vision should be tested and recorded at 10 feet, or even 5 feet, for the purpose of more accurate testing and as an indication on the patient's record that more than the usual care has been exercised for the visual determination.

NYSTAGMUS

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NYSTAGMUS means involuntary, more or less rhythmical to-and-fro movements of the eyes. In former times, nystagmus was considered as a pathologic sign occurring in, and characteristic of, certain diseases. Studies in the last few decades have revealed that the phenomenon of nystagmus is a physiologic one, and only the increased intensity of nystagmus or its occurrence under nonphysiologic conditions is specific for pathology.

Nystagmus is connected with four mechanisms: the fixation mechanism, the vestibular mechanism, the gaze mechanism, and the convergence mechanism. Therefore nystagmus could be divided into four forms:

1. Fixation nystagmus due to a disturbance of the fixation mechanism
2. Vestibular nystagmus due to a disturbance of the vestibular mechanism
3. Gaze nystagmus due to a disturbance of the gaze mechanism
4. Convergence nystagmus due to a disturbance of the convergence mechanism

The understanding of the mechanisms of the forms of nystagmus resulted, in recent years, in the possibility of eliminating nystagmus by treatment.

According to its appearance, nystagmus may be divided into pendular nystagmus and jerking nystagmus. Pendular nystagmus means that the two phases of the nystagmus movement have the same speed, while a jerking

nystagmus consists of a quick phase and a slow phase. Conventionally, the jerking nystagmus is denoted according to the direction of its quick phase; i.e., nystagmus with a slow phase to the left and a quick phase to the right is called right nystagmus. Pendular nystagmus is denoted according to its direction—horizontal, vertical, rotatory or circulatory nystagmus. Jerking nystagmus is divided according to the direction of the quick phase—right nystagmus, left nystagmus, up nystagmus, clockwise nystagmus, and so on.

Actually a so-called pendular nystagmus is pendular only in a certain part of the field of gaze; for instance, in a zone between 30 degrees to the left and 30 degrees to the right from the primary position. If the eyes look more to the right, the phase to the right becomes quicker and the nystagmus changes to a right nystagmus. In gaze to the left, a pendular nystagmus changes to a left nystagmus. The zone in which both phases have an equal speed is called the neutral zone of this nystagmus. In other words, the nystagmus is pendular only within this neutral zone and changes in gaze to the right to right nystagmus, in gaze to the left to left nystagmus. This neutral zone may be symmetrical to the primary position or it may be asymmetrical; for example, the right nystagmus starts at 10 degrees from the primary position, while the left nystagmus does not start until 40 degrees from the primary position. This is called asymmetrical pendular nystagmus.

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The jerking nystagmus also depends on the position of the eye. As a general rule, a nystagmus increases if the eye turns in the direction of the quick phase of the nystagmus, and decreases if the eye turns in the direction of the slow phase. For example, a right nystagmus increases in gaze to the right, and decreases or even disappears in gaze to the left. If the patient has a right nystagmus in gaze to the right, and a left nystagmus in gaze to the left, the zone between the two areas in which nystagmus appears is called the neutral zone. This neutral zone can again be either symmetrical to the primary position or asymmetrical. For instance, in gaze straight ahead there is right nystagmus. The right nystagmus increases in gaze to the right and disappears at 10 degrees to the left. If the eye is turned 40 degrees to the left, a left nystagmus appears. In this case the neutral zone lies between 10 degrees and 40 degrees to the left.

FIXATION NYSTAGMUS

Without fixation, the apparently quiet eye actually makes very fine to-and-fro movements; i.e., there is a kind of nystagmus, but its amplitude is so small that the movements can be visualized only with very high magnification. The fixation mechanism consists of impulses to the eye muscles which result in an increase of the frequency but decrease of the amplitude of the described physiologic nystagmus.

This fixation mechanism acts when three conditions are fulfilled: (1) presence of a well developed macula lutea; (2) formation of an image of an object with distinct contours on the macula; and (3) attention for the object. If these three conditions are met, the eye is fixed on the object by the described mechanism.

The most frequent form of fixation nystagmus is the so-called fixation nystagmus from infancy. This is often in-

accurately called optical nystagmus. It is seen with albinism, with scars of the cornea after ulceration in infancy (as in gonorrhea), with congenital absence of the iris, in congenital astigmatism, myopia or partial cataract. All these conditions have in common an indistinct image formation on the macula before the child is 4 months of age—before the macula has fully developed. If such a disturbance sets in after 4 months, i.e., after full development of the macula, no nystagmus can develop. In the exceptional case in which such a nystagmus is found without a visible cause, and with good vision, temporary damage in the first months of life, such as hemorrhage of the macula, may be assumed. The fixation nystagmus usually persists for life. Usually this nystagmus is pendular with symmetrical or asymmetrical location of the neutral zone. Rarely, there is only a right nystagmus and a left nystagmus but the eye appears quiet in the interposed neutral zone. If this neutral zone is asymmetrical, the nystagmus may be confused with one of the other forms of nystagmus.

Another form of fixation nystagmus which plays an important role in examination of refraction is the latent nystagmus. In binocular vision, the eyes stand still. When the right eye is occluded, both eyes have a jerking nystagmus to the left. When the left is occluded, both eyes have a jerking nystagmus to the right. When both eyes are occluded, there is no nystagmus. In other words, on occlusion of one eye, nystagmus appears to be in the direction of the open eye. This nystagmus, sometimes very strong, diminishes the visual acuity, and the test of vision gives paradoxical results; for instance, 20/100 in the right eye, 20/100 in the left eye, binocular 20/20. Since the jerking latent nystagmus to the side of the open eye decreases in gaze to the opposite

side, subjective refraction and even retinoscopy are possible if the open eye has been brought into maximal adduction, or looks over the nose. In this direction the vision may rise almost to normal.

The nystagmus of coal miners may be regarded as a kind of fixation nystagmus which develops because of lack of distinct contours in very poorly illuminated coal mines for many months or years.

An acquired pendular nystagmus is seen in multiple sclerosis and is pathognomonic for this disease. In contrast to the nystagmus from infancy, the acquired pendular nystagmus results in oscillopsia, which means that the patient sees a seeming to-and-fro movement of objects.

VESTIBULAR NYSTAGMUS

If the head is rotated involuntarily to one side, the labyrinth causes a compensatory movement of the eyes in the opposite direction so that the eyes more or less stay at the same place in relation to the surroundings. In more extensive head rotations this compensatory deviation of the eyes is replaced by a series of compensatory deviations alternating with quick returns of the eyes to the original position, resulting in a nystagmus of the eyes. This is the only physiologic vestibular nystagmus. If the labyrinth is inadequately stimulated, such as by rotation in a revolving chair or by pouring cold or hot water into one ear, an unphysiologic nystagmus appears. If one labyrinth or its connection with the central nervous system is irritated or damaged, a pathologic vestibular nystagmus results.

GAZE NYSTAGMUS

If, due to a lesion in the motor apparatus, gaze to the right brings the eye more or less to the right end position but this position is not maintained, repeated new impulses have to be sent to

the eyes. Thus a nystagmus to the right results. The gaze nystagmus may be symmetrical; that means, it may appear in gaze to the right, in gaze to the left, and in gaze upwards at the same distance from the primary position. Such a symmetrical gaze nystagmus is in most cases due to multiple sclerosis. If the gaze nystagmus is asymmetrical; for example, the right nystagmus is much stronger than the left nystagmus, a lesion in the brain stem is indicated. Symmetrical gaze nystagmus is indicative of a certain disease, while asymmetrical gaze nystagmus suggests a certain location of the lesion.

CONVERGENCE NYSTAGMUS

To this form belong rare phenomena like the voluntary nystagmus consisting of a tremor of the convergence, and the so-called retraction nystagmus in which the eyes jump backwards into the orbit; the latter sign is indicative of a lesion in the midbrain.

Fixation nystagmus can be differentiated from the other forms by use of the optokinetic drum, a drum covered with alternating black and white stripes which is rotated in front of the eye. The normal eye shows an optokinetic nystagmus as it looks at the rotating drum, because the eye is fixed on a moving stripe for a time and then jumps back to another stripe. In fixation nystagmus, a nystagmus due to a disturbed fixation, the eyes cannot be fixed on a stripe, and therefore the normal optokinetic nystagmus is absent. In the presence of other forms of nystagmus, such as vestibular nystagmus, the optokinetic nystagmus is elicited normally.

THERAPY

In cases in which the nystagmus is present in the primary position but absent in another position, the nystagmus in the primary position can be eliminated by surgical correction. Let us assume that nystagmus is present in the pri-

mary position but is small or even absent if the patient turns his eyes 5 mm. to the left (corresponding to 40 prism diopters). This position is called optimum position. In the operation, both eyes are rotated surgically just 5 mm. to the right, i.e., opposite to the optimum position. This can be done by 5 mm. recession and 5 mm. resection of the corresponding muscles. Altogether, all four horizontal muscles are operated upon. When the eyes have been rotated surgically 5 mm. to the right, a left impulse to the extent of 5 mm.

brings the eyes back to the primary position. Since the eyes do not show nystagmus on a left impulse of 5 mm., the eyes are quiet in the primary position. This operation not only produces a very good cosmetic result, but elimination of the nystagmus may cause an enormous improvement of the vision in the primary position. It is self-evident that such an operation is permitted only if the nystagmus is stationary, and improvement, spontaneous or due to treatment of the causative disease, cannot be expected.

In Memoriam



JEAN STOCKS ROBINSON

September 7, 1912 — November 30, 1957

Jean Robinson was vitally interested in orthoptics and the American Association of Orthoptic Technicians, working unstintingly for the benefit of her patients and for the good of the Association. She served as president and as secretary-treasurer of the American Association of Orthoptic Technicians, worked tirelessly on many committees, contributed to professional journals and helped examine candidates for certification.

She was a person admired by all who met her and loved by those who knew her.

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THE NEED FOR MORE ORTHOPTIC TECHNICIANS

Workers in all types of medical auxiliaries are in short supply. Positions are available for medical record librarians, medical record technicians, medical technologists, occupational therapists, orthoptic technicians, and others upon whom doctors of medicine necessarily rely, and there are not enough trained individuals to fill them. Even if all paramedical schools were filled to capacity each year, an insufficient number of applicants would be available. Yet most schools do not have a complete enrollment. The Council on Medical Education and Hospitals of the American Medical Association reports that approved schools for medical re-

cord librarians have a capacity for 262 students, and in 1956 there were 145 students enrolled. With a student capacity of 4,796, the occupancy rate for schools for medical technologists in 1956 was only 61 per cent.

A similar situation exists in the field of orthoptics. In spite of the fact that many positions available to orthoptic technicians remain unfilled, the number of students certified each year by the American Orthoptic Council is barely large enough to replace technicians who retire for one reason or another. In part, this is because existing facilities for training technicians are limited; but even these limited facilities are not be-

ing used to capacity. While it is recognized that finding more student orthoptic technicians would be worthless without facilities for training them, the fact that our training centers are not filled to capacity makes our primary problem one of recruitment.

The American Orthoptic Council has approached the problem by publishing a pamphlet entitled "A Profession in Orthoptics," copies of which have been distributed to vocational guidance counselors in every junior college in the United States and to every member of the American Personnel and Guidance Association. Numerous libraries, high schools, and interested individuals have requested and received the pamphlet. Undoubtedly, this distribution has been successful in a small degree and will be more productive as time passes.*

To solve the problem of recruitment effectively, there must be a coordinated approach. Orthoptic technicians, themselves, can help by word of mouth advertising. Every technician should make it her job to inform young women of her acquaintance of the advantages of orthoptics as a profession and to aid them in seeking training. Ophthalmologists may find opportunities to acquaint local groups such as high school science clubs and PTA organizations with this field of endeavor.

The problem faced by the ophthalmologist who wishes to employ a technician but can find none is a real one. A technician does not often wander in to an ophthalmologist's office seeking employment; most of us cannot wait for this to happen. The American Association of Orthoptic Technicians conducts a placement bureau and occasionally a

technician may be found by applying to the bureau. Supply and demand is such, however, that last year only five ophthalmologists secured the services of a technician through the efforts of the bureau.

At the present time, the surest way for an ophthalmologist to obtain the services of an orthoptic technician is to find a qualified young woman in his locality and help her to obtain training. He may send her to one of the centers offering complete didactic and practical training or he may wish to have her enroll in the two month basic course in orthoptics which is conducted by the American Orthoptic Council each summer. Students who have satisfactorily completed the basic course are assisted by the Council in obtaining ten months of practical training leading to the examination for certification. An ophthalmologist who assists a student to obtain training may reasonably expect to utilize her services when she receives her certificate. Financial arrangements between the ophthalmologist and the student are matters of individual consideration. Scholarships to aid students needing help are available through the Delta Gamma Fraternity and applications for such scholarships may be made to Mrs. H. L. Marchant, 4006 Virgilia, Chevy Chase 15, Maryland.

Nowadays, the public is fully aware of modern medical treatment and more and more people are making use of regular care by an ophthalmologist. There have been great technological advances in ophthalmology, and each advance places greater emphasis on the role played by medical auxiliaries. Ophthalmologists need well-trained technicians to assist them and in generous quantity. To help supply technicians to fill this need is the responsibility of all of us.

EDMOND L. COOPER, M.D.

*The pamphlet describes the work of the orthoptic technician, outlines the qualifications desirable in a student, discusses training costs and prospective earnings, and lists training centers. It is available to anyone on application to the office of the Secretary of the Council.

ORTHOPTIC COURSE MOVED TO ANN ARBOR

During the last Academy meeting the American Orthoptic Council decided that the basic course in orthoptics for technicians should be given in a new location during the summer of 1958. Although the course has been given with great success under the direction of Dr. Hermann M. Burian, with the enthusiastic cooperation of Dr. Alson Braley and other members of his staff at Iowa City, the Council felt that, in fairness to them, the load should be shifted. The American Orthoptic Council and the American Association of Orthoptic Technicians wish to thank the entire group at the University of Iowa.

In searching for a new location, the Council decided that a university community would be the best site, and chose the University of Michigan at Ann Arbor.

Plans are well under way for the 1958

summer course, which will be given as an official summer session course of the University of Michigan. Regular registration in the University has been arranged including room and meals in the dormitory adjacent to the University Hospital, where the classes will be held. Students will be eligible for University Health Service facilities and use of the Michigan League of Women's athletic facilities, which include the new women's swimming pool, which is close to the women's dormitories. The practical demonstrations will be given in several locations, utilizing facilities in neighboring Detroit.

For further information with regard to the course, please contact John W. Henderson, M.D., Room 6155, Out-patient Building, University Hospital, Ann Arbor, Mich.

J. W. H.

AMERICAN ORTHOPTIC COUNCIL — 1958

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ABSTRACTS OF OPHTHALMIC LITERATURE

These abstracts were made possible by the Assistant Editors, Dorothy Reimer and Marjorie Snell, with the help of the following Committee:

Margaret Crush
Mary Hefferman
Helen Hellebo

CLASSIFICATION

1. Accommodation
2. Amblyopia
3. Anomalous retinal correspondence
4. Diplopia
5. Divergent deviations
6. Esotropia
7. Fusion
8. Instruments and devices
9. Miscellaneous
10. Paralysis
11. Physiology
12. Refractive error
13. Stereopsis
14. Surgery
15. Torsion
16. Vergences
17. Vertical deviations
18. Vision

1-1

Breinin, Goodwin M.: *Relationship between accommodation and convergence*, Tr. Am. Acad. Ophth., 61: 375-382 (May-June) 1957.

In order to treat accommodative esotropia, the physician must first have a clear understanding of the mechanisms underlying vergence movements. Dr. Breinin discusses each type of vergence innervation as classified by Maddox: proximal, tonic, accommodative, and fusional.

Proximal vergence is not clearly understood, and it is not known "whether this is an independent entity or merely a psychological facilitation of other types of convergence innervation." Healthy persons have little proximal vergence;

patients with strabismus may have considerably more.

"Tonic vergence is that basic innervation which carries the eyes to the dissociated position for distance fixation when accommodation and fusion are suspended. It is the resultant of many primitive reflex and quasi-reflex mechanisms, such as cortical, labyrinthine, proprioceptive and visual, and of unknown sources which funnel into the opposed channels of convergence and divergence, thus modifying the basic anatomical position of the eyes." Tonic esotropia is not considered in this paper but it is clear that the excessive tonic convergence in infancy forms the background upon which other factors may act to predispose the child to a convergent angle of the visual axes. Failure of the corrective optomotor reflexes allows the deviation to become manifest.

The accommodative mechanism does not usually develop fully until a child is about two years old. Then the eyes must accommodate to see close objects clearly and the eyes must converge in order to see them singly with binocular vision. Thus the close association of accommodation and convergence is established. "It cannot be definitely stated to what extent this bond is innate or learned, but the evidence favors the view of an inherent association which, perhaps, may be modified by experience through trial and error."

The author discusses the concept of the meter angle and points out the importance of evaluating the actual angle of convergence required for binocular fixation. The measurement of the interpupillary distance (P.D.) is essential to determine the required angular convergence in prism diopters. "The angle of convergence exists between two eyes, so it is more meaningful to speak of the two eyes having a convergence of one meter angle rather than of each eye converging one meter angle." By multiplying the interpupillary distance in centimeters by the dioptric distance of the object of regard, the physician can obtain the actual angle of convergence in prism diopters.

Now the subject must also accommodate to see clearly. A patient with emmetropia must accommodate one diopter and converge six prism diopters at a distance of one meter if his interpupillary distance is 6 cm. If his interpupil-

lary distance is 5 cm., he must converge 5 prism diopters. At a distance of one-third meter, three diopters of accommodation are needed and the respective convergence would be 18 diopters (6 cm. P.D.) and 15 diopters (5 cm. P.D.). The number of meter angles converged would be the same in each case.

"Accommodative effort is the factor associated with convergence—the resultant is termed accommodative convergence. The amount of accommodative effort elicited by a stimulus is the key to the amount of convergence effected, and not the stimulus per se, or the amount of accommodation actually effected."

The amount of accommodative convergence is roughly proportional to the effort. "Hill has reported a useful formulation which states that the approximate amount of accommodative convergence in prism diopters elicited by an object is obtained by multiplying the interpupillary distance by the dioptric measure of the accommodation necessary for a given distance of the object. . . . The hypermetropic subject then will overconverge for a given distance, but through the corrective fusional reflex he may retain binocular single vision." This will result in an esophoria; however an esotropia will be manifest if the fusional reflex is inadequate.

Dr. Breinin discusses the term accommodative convergence-accommodation ratio in detail and concludes that although there is an accommodation convergence relationship there is not necessarily a ratio.

Fusional vergence must be exerted to bring the eyes from a fusion-free position into exact alignment with the fixation point. "The stimulus to fusional vergence is retinal disparity of images. . . . In accommodative esotropia the eyes are in an overconvergent position. . . ." and the corrective fusion movement to obtain binocular single vision is one of divergence. "This is termed *relative fusional divergence*. . . . It is the presence and amplitude of relative fusional divergence which determines whether a given case of accommodative esotropia can be restored to binocular single vision, i.e., to esophoria or orthophoria." It may be demonstrated by (1) finding the weakest plus-lens correction which will permit clear binocular single vision, (2) the base in blur test (addition of base in prisms while the eye being tested fixates a fine symbol at a distance of one-third meter until blurring occurs), and (3) the use of a haploscope.

A knowledge of the roles played by lenses and fusion training is essential in the treatment of accommodative esotropia.

1-2

Hill, Robert V.: *The hyperbolas of accommodation and convergence*, A.M.A. Arch. Ophth., 57: 259-265 (Feb.) 1957.

The author presents a mathematical analysis of the normal functions and interrelationships of accommodation and convergence. By "using constant units of measurement it is impossible to express in a simple equation the coordinate roles of an algebraic function (accommodation) and a transcendental or nonalgebraic function (convergence)." Dr. Hill changes the transcendental function of convergence to an algebraic one by using a noncircular and inconstant unit of measurement—the prism diopter. When this is done, accommodation and convergence have identical mathematical solutions (equilateral hyperbolas). Mathematical analysis of accommodation and convergence relative to their parametric variable, fixation or focal distance, is then possible.

2-1

Catford, G. V.: *Amblyopia—a comparison between distance and near vision*, Brit. J. Ophth., 40:633-635 (Oct.) 1956.

A comparison has been made of distance and near vision in the amblyopic eyes of 50 applicants for the R.A.F. The R.A.F. near point rule was used for assessing near vision. Fifty-four per cent were found to have near vision equivalent to N 12 or better.

2-2

Hauser, Paul J., and Burian, Hermann M.: *Fixation patterns in strabismic amblyopia*, A.M.A. Arch. Ophth., 57:254-258 (Feb.) 1957.

This paper presents results of tests done in the method of Brock and Givner, who showed that many amblyopic eyes fixated eccentrically.

The methods (using afterimage localization) and clinical materials used are described in detail. The authors ran the tests on twenty-five patients and their results showed that the test of Brock and Givner did not give evidence that the majority of patients fixated eccentrically with their amblyopic eye. They did find that some patients required a stronger stimulus in order to fixate centrally with the amblyopic eye. Anomalous retinal correspondence and wandering attention placed limitations on the test.

2-3

Kupfer, Carl: *Treatment of amblyopia ex anopsia in adults*, Am. J. Ophth., 43:918-922 (June) 1957.

"The purpose of this paper is to determine whether an adult with an amblyopia resulting from a childhood squint could be taught to fix centrally with his poor eye, whether learning central fixation would enable the visual acuity to improve, and whether this increased acuity would be maintained after treatment was completed."

The criterion for choosing the patients was only their desire to undergo hospitalization to see if the vision in the poor eye could be improved. Seven patients were selected.

The good eye was padded, using two eye pads and adhesive tape, and every four or five days this occlusion was changed in a darkened room with both eyes closed. After one week the visual acuity of the amblyopic eye was checked. Except for one patient, there was no significant improvement. Then the problem was to teach the other six to fixate with the true macula; this was accomplished as follows:

"1. Fixation on an ophthalmoscope light during simultaneous direct observation by the examiner. . . . the optical disc of the pantoscope was set for macula examination and the diameter of the light beam controlled with the iris diaphragm. The patient was told in which direction to turn his eye for macula fixation and told when the light was centered on the macula. He then attempted to correlate firstly, the difference in the brightness of light which he experienced when the light was directed straight through to the macula as compared to the sensation of brightness experienced when the light was thrown obliquely onto the peripheral retina and, secondly, to assess the direction and amount of movement which his eye had to make to fix the ophthalmoscope light; this the patient found difficult but made easier by the sensation of relative adduction and abduction of the padded eye."

2. When the patient could fixate a light with his macula he wore a pinhole lens and fixated the light held up to distances of five feet and large letters up to distances of one or two feet away. Patients stated that the pinhole lens helped them to hold macular fixation more easily.

"3. The central field of the amblyopic eye was mapped periodically on the tangent screen. The relationship between the point of fixation, the blindspot and any scotomas gave some indication of whether there was central fixation or not."

The results indicated that the individual's intelligence and motivation are of utmost importance in regaining vision in an amblyopic eye. Of the seven patients studied, one patient with an initial visual acuity of hand movements improved to 20/25; another with hand movements, to 20/40; two patients with an initial visual acuity of 20/200, to 20/30; one patient with faculative amblyopia improved from 20/70 to 20/20. Two patients with visual acuities of 4/200 and 5/200 improved only to 20/200.

Central macular fixation must be obtained before any improvement in visual acuity is to be expected. In the patients showing improvement, the improvement was maintained when the patients were retested 24 weeks after discharge. Patching was discontinued during this time and no further advice was given as to the use of the eyes.

3-1

Swan, Kenneth C., and Wahlgren, Ruth E.: *Anaglyphic phenomena in anomalous correspondence*, A.M.A. Arch. Ophth., 57:842-845 (June) 1957.

Patients were given red and green filters which were balanced so that the light stimuli to the eyes were equal. They then looked at a brightly lighted, unmarked white screen three to four feet away.

Persons with normal binocular vision see the screen a mixed red and green or a dominance of one and then the other. Persons with esotropia or exotropia, normal correspondence and good vision describe a modification of the same. Patients with amblyopia see it monocularly.

To a person with an esotropia and harmonious anomalous correspondence, with the red filter over the right and fixing eye and the green filter over the left and deviating eye, the central area and all the left half of the screen appear red. Most of the right side appears green but there is no sharp dividing line.

In exotropia and harmonious anomalous correspondence with the red filter over the fixing eye (right) and the green over the left eye, the fields appear uncrossed. The right half is red and the left half green.

Persons with vertical deviations were also tried and if the deviations were anomalous, they too had similar crossing of fields.

"The phenomenon is best explained on the basis of predominance of the macular region of each eye over the abnormally corresponding peripheral retinal areas in the other eye when dissimilar stimuli of equal intensity are presented to these regions."

6-1

Bayard, Walter L.: *Permanent esotropia induced by cycloplegia*, Am. J. Ophth., 43:119-120 (Jan.) 1957.

This paper reports a case of permanent esotropia following atropine cycloplegia in a nine-year-old girl. The only history was of an esotropia and diplopia for a period of four to six hours a year before. Both the esotropia and diplopia disappeared spontaneously. She had 20/20 vision in each eye, four prism diopters of exophoria for near, and first, second, and third grade fusion on the troposcope. One drop of one per cent atropine was instilled in each eye every five minutes for three doses. One hour later the patient showed a twenty degree left esotropia and homonymous diplopia. Refraction revealed a +1.25 diopters sphere in each eye. Pilocarpine was instilled in each eye. The esotropia remained the same. The patient was given two per cent pilocarpine three times a day in each eye and given full correction. Intensive visual training was given for six weeks. Findings remained unchanged. The patient was seen at irregular intervals for the next three years and findings remained the same.

Three years after onset a bilateral resection of the lateral recti and a recession of the left medial rectus were performed. Six weeks after the operation there was an esophoria of thirty diopters for distance and two diopters for near. Binocular vision was unchanged from the initial findings.

6-2

Costenbader, Frank D.: *Principles of treatment*. In *Symposium: Accommodative Esotropia*, Tr. Am. Acad. Ophth., 61:390-394 (May-June) 1957.

Treatment for accommodative esotropia should be instituted early in order to maintain an opportunity for fusion and to prevent secondary neuromuscular changes which may cause constant deviation. This treatment re-establishes binocular alignment and binocular vision by the use of glasses, bifocals, miotics, and at times surgical intervention. When the child is cooperative, orthoptic training is used to improve fusion and fusional amplitudes and particularly relative fusional divergence.

First, visual acuity must be regained for patients with amblyopia by means of occlusion. Emphasis is placed on obtaining good visual acuity while the child is viewing the full chart rather than isolated symbols and on ability to maintain accurate foveal fixation. Anisometropia and organic damage make the regaining of visual acuity difficult.

Second, binocular alignment of the visual axes must be regained and maintained at the same time effort is being made to improve visual acuity. This is accomplished by the use of glasses, unifocal or bifocal; miotics; surgical intervention; or combinations of these methods. For patients with a normal A/C ratio, spectacles of sufficient strength to relieve accommodation and excessive convergence should be prescribed. Orthoptic treatment should be given to improve relative fusional divergence. If the esotropia is totally eliminated for distance but some residual esotropia remains for near fixation, a bifocal segment in the lens or miotics may be helpful. If a combined accommodative and nonaccommodative esotropia is present after the child has worn full hyperopic correction, an operation should be done to correct only the nonaccommodative element.

In accommodative esotropia with an abnormal A/C ratio, both the esotropia for distance and the hyperopia are small, but in most cases there is a large esotropia at 13 inches when the patient fixates a small symbol. Generally the residual esotropia for near can be eliminated by adding a +3.00 sphere. When such is the case, a large (38 mm.) Ultex A type of bifocal should be prescribed. If the esotropia is not completely relieved by the bifocals, one drop of Floropryl, .025 per cent, in each eye daily, may eliminate the residual esotropia.

Dr. Costenbader has listed several personal opinions on surgical intervention in accommodative esotropia.

After good visual acuity has been obtained and proper alignment of the visual axes has been accomplished by some method, orthoptic treatment may be instituted to overcome suppression, encourage diplopia, increase fusional amplitudes and develop relative fusional divergence. As relative fusional divergence develops, the strength of the glasses may be gradually reduced and finally eliminated if the hyperopia is not more than +4.00 sphere.

To find out the least hyperopic correction necessary to maintain alignment of the visual axes distance and near while the patient is accommodating, the following procedure is suggested:

Full cycloplegic correction is placed in the trial frame and the patient maintains 20/30 visual acuity for distance. The hyperopic correction is simultaneously reduced .50 in each eye and cover-uncover test performed to see that binocular vision is maintained. This procedure is continued until the weakest strength allowing binocular single vision is reached. Similar examination for near fixation is done starting with full correction and +3.00 add if necessary.

The eyes should be examined for minimal correction at least every six months and glasses reduced if possible.

Prognosis in accommodative esotropia is good if the A/C relationship is good, treatment is started soon after onset, good follow-up is maintained and significant amblyopia or organic visual loss is not present.

6-3

Gill, Elbyrne G.: *Management of crossed eyes based on experience in 450 patients*, EENT Monthly, 36:25-28 (Jan.) 1957.

Treatment should be instituted as soon as a squint is observed, regardless of the age of the patient. The author feels that if a physiologic result is to be obtained, medical treatment (atropinization of the eyes, occlusion of the fixating eye and correction of the refractive error) and probably surgical treatment must be carried out before age three. If medical treatment is not successful in six to twelve months, then surgical correction is indicated regardless of the age of the patient. Dr. Gill feels that medical treatment and orthoptic exercises give best results in patients with less than fifteen prism diopters of squint. Orthoptic training should be delayed until after age four and is a valuable aid in preoperative and postoperative treatment.

Tests for diagnosis are discussed. A classification guide for strabismus is listed, and surgical principles and type of anesthesia are discussed.

6-4

Schlossman, Abraham: *Accommodative esotropia with abnormal accommodation-convergent ratio*, EENT Monthly 36:48-51 (Jan.) 1957.

Twenty-five percent of all cases of strabismus are classed as accommodative esotropia. A number of patients have a residual esotropia for near even when wearing full cycloplegic corrections. In such cases further treatment steps must be taken. In the average case adequate nonsurgical methods are available—local DFP instillation and/or orthoptic training.

Another group of patients remains in which the A/C ratio is very abnormal. Treatment of these patients is difficult because the above methods are usually inadequate to control this squint and an operation may result in over-correction.

Very few patients with accommodative esotropia have had surgical treatment during the

past decade. The author feels that as the child matures and the accommodation becomes weaker, the esotropia for near gradually decreases. He feels this condition should be treated conservatively lest the patient develop a consecutive exotropia for distance later in life.

6-5

Swan, Kenneth C.: *Classification and diagnosis*. In *Symposium: Accommodative Esotropia*, Tr. Am. Acad. Ophth., 61:383-389 (May-June) 1957.

The general features of accommodative esotropia are described in this paper. In early accommodative esotropia there is no "basic deviation" which is typical. There is only an abnormal degree of convergence induced by excessive accommodative effort. It is usually noticed first when the child is two years old or slightly older; however it may be found as early as eighteen months or as late as five years. Most of the time the deviation is present initially only when the child is tired, then over a period of time the deviation tends to become more persistent for distant and near fixation. Occasionally the esotropia appears suddenly and persists. "Hypermetropia of more than average amount is the most typical finding associated with accommodative esotropia." Anisometropia and astigmatism of high magnitude are more frequent than in a comparable group of children with normal binocular single vision. Amblyopia is more frequently found than in most types of strabismus, but a marked amblyopia with variable or eccentric fixation is rare. Anomalous retinal correspondence is uncommon due to the variable deviation and relatively late onset, but it may be found in neglected cases of early onset.

In addition to the general features of accommodative esotropia mentioned above, Dr. Swan has subdivided and discussed the cases according to differences in course and treatment:

- "1. *Typical Accommodative Esotropia*
Deviation is due, primarily, to uncorrected hypermetropic refractive error. Normal accommodation-convergence relationship is restored by glasses.
- "2. *Atypical Type of Accommodative Esotropia with Residual Overconvergence in Near Vision*
The average refractive error is less than that in typical cases. Single vision glasses do not restore normal accommodation-convergence relationship.
- "3. *Accommodative Type of Esotropia Due to Uncorrected Hypermetropia in One Eye,*

and Sensory Obstacles of Fusion, e.g., Impaired Vision, in the Other

"4. Mixed Accommodative and Nonaccommodative Types

- a. Basic esotropia with an accommodative component
- b. Neglected accommodative esotropia with sensory and motor adaptive mechanisms."

All types of accommodative esotropia will usually develop into a constant esotropia if untreated, and although there are reports of occasional spontaneous cures in untreated accommodative esotropia, the course is usually not favorable. A consecutive exotropia may develop by the time adolescence is reached. This condition is more likely to occur in those patients who did not develop a normal type of binocular single vision in early childhood. They include patients with "intractable unilateral amblyopia, marked anisometropia or poor vision, of any origin, in one eye." Children with six or more diopters of hypermetropia also tend to develop consecutive exotropia.

7-1

Jampolsky, Arthur; Flom, Bernice; and Freid, Alan N.: *Fixation disparity in relation to heterophoria*, Am. J. Ophth., 43: 97-106 (Jan.) 1957.

The authors describe a study done to determine the type and amount of fixation disparity existing under approximately natural visual conditions, and also to determine the type of relationship existing between fixation disparity and horizontal heterophorias.

A group of fifty-seven subjects was selected. All had corrected visual acuity of 20/20 in each eye and had no history of strabismus (past or present), or visual training. Procedure and apparatus for determining heterophoria and fixation disparity are described.

The interpretation of the results of the study indicates that for distant fixation, large values of esophoria are associated with large values of convergent fixation disparity, but for exophoria there is little or no relationship between the degree of exophoria and fixation disparity. The relationship between fixation disparity and heterophoria is different in esophoria from that in exophoria. For near fixation, the relationship between fixation disparity and heterophoria is apparently the same for both esophoria and exophoria; the data indicate that increasing amounts of esophoria and exophoria are associated respectively with increasing amounts of convergent and divergent fixation disparity.

8-1

Askovitz, S. I.: *A device for the demonstration and analysis of ocular rotations*, Am. J. Ophth., 43:765-766 (May) 1957.

An apparatus is described which is useful for demonstrating to students of ophthalmology combinations of ocular rotations. It is also useful in investigating true and false torsion.

The device was constructed of two government surplus navigating compasses which were stripped of all unnecessary attachments and mounted together. Either "eye" can be rotated independently about a horizontal, vertical, or sagittal axis. Each rotation can be read to the nearest degree of arc on the corresponding scale of the instrument.

Versions, vergences and torsions are readily clarified by setting the two "eyes" accordingly.

8-2

Berens, Conrad; Brackett, Vivian, and Taylor, Evelyn B.: *Duochrome television eye exerciser*, Am. J. Ophth., 43:771-772 (May) 1957.

The Duochrome Television Eye Exerciser consists of a red and green plastic cover which is hung in front of the television screen and is used with red and green spectacles and prism bars. The red and green strips are mounted on a sheet of thin transparent plastic. A circular opening 10 cm. in diameter, eccentrically placed, extends equally into the red and green sections.

The only part of the screen which can be seen with each eye simultaneously is this central opening. If two round openings are seen, a horizontal prism bar is used and, if needed, a vertical bar, until the circles are superimposed. When fusion is stable, converging, diverging and supraverging exercises may be carried out as prescribed.

In cases of amblyopia or suppression, the cover should be placed so that the red portion covers the upper half of the screen. The red lens should then be worn before the suppressing or amblyopic eye.

8-3

Costenbader, Frank D.: *Fit-over frame*, Tr. Am. Acad. Ophth., 61:224 (March-April) 1957.

The "fit-over cell" is devised to clip on the patient's own glasses and simplifies the use of the Maddox rod, the pinhole, the blank and other auxiliary lenses. It is especially useful

for examining children. "Most trial frames do not place lenses as accurately before the eyes as do the child's own glasses. Thus if the fit-over cell is used on the child's glasses, the lenses are most accurately centered."

8-4

Costenbader, Frank D.: *The improved accommodometer*, Tr. Am. Acad. Ophth., 61:225 (March-April) 1957.

The accommodometer is described in detail. Its three primary uses are (1) accurate testing of the near point of accommodation, (2) estimating the visual acuity at 13 inches, and (3) determining the weakest hyperopic correction essential for good vision in accommodative esotropia.

8-5

Fink, Walter H., and Fink, Robert J.: *Fixator for the version test*, Tr. Am. Acad. Ophth., 61:415 (May-June) 1957.

A white sphere, one inch in diameter, is attached to an arm which, in turn, is attached to a handle. The arm may be rotated in any direction and the white sphere may be moved by sliding it on the arm.

The handle is held by the patient fifteen inches in front of himself and he fixates the white ball as it is moved into the cardinal positions. The examiner then has both hands free for measuring the squint in the various fields of gaze.

8-6

Graham, P. A., and Naylor, E. J.: *A photographic method of measuring the angle of squint*, Brit. J. Ophth., 41:425-433 (July) 1957.

The authors point out that the disadvantage of measuring the angle of squint on the major amblyoscope is that although the fixation object is optically at infinity, the known proximity of the apparatus may stimulate convergence. An attempt has been made to overcome this difficulty by assessing the angle of squint by photographic means. This method also has the advantage of providing a permanent record.

The apparatus and method used are described in detail (camera, film, patient's distance from the camera and fixation target, the mathematical reasoning for the method used, etc.).

Fifteen children were examined on admission to the hospital for surgical treatment of a convergent squint. The results of these examinations are tabulated.

Detailed discussion is given regarding possible sources of error for this method of measuring a squint: errors due to theoretical assumption regarding the optical constants of the eye and those due to inaccuracies of measurement in practice.

9-1

Burnside, Ronald M., and Langley, Charlotte: *Screening test for aniseikonia*, Am. J. Ophth., 43:620 (April, pt. I) 1957.

The authors describe a method using the major amblyoscope for screening patients with suspected aniseikonia to determine whether or not a more complete evaluation on the eikonometer is indicated. The pair of targets used is described. Where a major amblyoscope is not available, the same targets can be transferred to a card for use in the stereoscope.

9-2

Lyle, T. Keith, and Foley, Jill: *Prognosis in cases of strabismus with special reference to orthoptic treatment*, Brit. J. Ophth., 41:129-152 (March) 1957.

The authors state that experience with numerous patients with strabismus has taught that binocular single vision can be restored in many cases if correct treatment is given, while in other cases single binocular vision cannot be attained no matter what treatment is given. Treatment varies according to the type of strabismus. A careful study of the history of the patient and a thorough examination will determine what sort of treatment is needed, and give a fairly accurate prognosis as to whether or not binocular single vision will be attained.

The types of strabismus are (1) nonparalytic accommodative convergent strabismus, (2) nonparalytic nonaccommodative convergent strabismus, (3) constant divergent strabismus, (4) congenital paralytic strabismus.

These groups are subdivided further with regard to amblyopia and retinal correspondence. Stress is laid upon careful history-taking especially with regard to age and mode of onset and heredity.

Orthoptic treatment may be valuable in all types of accommodative strabismus, although correction of the refractive error suffices in some cases. Orthoptic treatment is valuable also for patients with late onset of nonaccommodative convergent strabismus to ensure equal visual acuity, absence of suppression, etc. Orthoptic treatment is helpful in divergent strabismus and, in some cases of intermittent divergent squint, orthoptic training

alone may suffice. Orthoptic training is valueless for treating congenital paralytic strabismus, but orthoptic methods of diagnosis in these cases are desirable.

In conclusion the authors state that the restriction of orthoptic treatment to cases in which there is a reasonable chance of recovery of the binocular function would allow more time to be spent on patients who deserve treatment and in detailed investigation and diagnosis of patients who require surgical treatment.

9-3

Schlossman, Abraham: *Summary of the value of orthoptic training in heterotropia*, EENT Monthly, 36:175 (March) 1957.

The foremost value of orthoptic training lies in the treatment of amblyopia. Along with treatment by occlusion the patient should be required to utilize vision in the poor eye by simple exercises such as filling in o's, dotting i's, and putting toothpicks into small holes. After vision has been improved, the patient should be taught how to maintain fixation with the amblyopic eye.

Orthoptic training is also a valuable treatment for strabismus by teaching the patient how to dissociate accommodation and convergence. It is the main form of treatment in convergence insufficiency.

Preoperative and postoperative orthoptic training is valuable in helping to consolidate and maintain a good functional result.

Orthoptic training is the treatment of choice in heterophoria with symptoms.

Orthoptic training is of doubtful value in the presence of anomalous retinal correspondence, therefore the operative approach is the chief form of treatment in this condition. Following surgical treatment, orthoptics may be valuable in helping to establish normal retinal correspondence.

9-4

Schlossman, Abraham: *Heredity in strabismus and glaucoma*, EENT Monthly, 36:301 (May) 1957.

Forty-seven and one-half per cent of all patients with squint belong to families containing two or more members with this condition. Certain clues point to an inherited innervational factor and others to an inherited anatomic factor but the exact factor is unknown.

Clues which point to a neurologic nature of strabismus are nystagmus, mental retardation, stuttering and left-handedness.

Clues which point to an anatomic factor are variations in size, thickness and insertions of the muscles.

Clinically the direction of the squint is usually the same in all members of a family with strabismus.

Since it is known that heredity plays such an important part in strabismus, as many children as possible in the same family should be examined in order to discover possible early amblyopia.

In the surgical management, a study of the end results of surgical treatment of a member of the family in which a similar squint prevails offers a safe guide in planning operations on subsequent members.

9-5

Trevor-Roper, P. D.: *The treatment of squint by exercises*, Brit. J. Phys. Med., 20:58-62 (March) 1957.

Eye exercises have an important part in the treatment of squint, but their major importance is in aiding the eyes to perform in unison. Thus the various so-called sight-improving exercises have no clinical or scientific basis.

The etiology of squint, the clinical picture with regard to diplopia and amblyopia, the treatment of concomitant squint, the importance of a good refraction, occlusion, and orthoptic training with a view to abolishing suppression and developing fusion and stereopsis, a few means of treatment such as the amblyoscope and cheiroscope, and surgical procedures associated with them are discussed. The author believes that, in adults, surgical treatment for cosmetic reasons is ample justification for suggesting an operation.

In conclusion it is stated that a squint is a disfigurement that affects the personality of the sufferer, and that in alleviating this, when possible, the orthoptist has a rewarding task.

10-1

Jain, N. S.: *Synkinetic lid retraction*, Brit. J. Ophth., 41:247-253 (April) 1957.

Abnormal lid retraction of the upper eyelid in congenital ptosis and acquired oculomotor ophthalmoplegia is not as uncommon as previously thought. Dr. Jain describes nine patients, giving the age at onset, sex, cause of ophthalmoplegia, and manifestations.

Dr. Jain states that the paradoxical lid retraction on occlusion of the sound eye was congenital in two cases and acquired in two more, due to typhoid and head injury. Fuchs' phenomenon of retraction on attempted adduc-

tion was present in three cases due to subarachnoid hemorrhage, typhoid, and head injury, in one case which was congenital, and in one after syphilis. The pseudo-Graefe phenomenon was not seen as a congenital lesion in this series, but occurred in two cases after subarachnoid hemorrhage and head injury. The Marcus Gunn phenomenon, Duane's retraction syndrome, and myasthenia gravis were seen also. In one case Fuchs' phenomenon and the pseudo-Graefe phenomenon were combined with paradoxical lid retraction on occlusion of the sound eye; this rare combination does not seem to have been reported previously.

10-2

Lincoff, Harvey A., and Cogan, David G.: *Unilateral headache and oculomotor paralysis not caused by aneurysm*, A.M.A. Arch. Ophth., 57:181-189 (Feb.) 1957.

Aneurysms of the internal carotid artery cause the syndrome of unilateral frontal headache and oculomotor paralysis. These symptoms occur only rarely with aneurysms elsewhere. There is prevalent belief that unilateral headache and oculomotor palsy are almost pathognomonic of aneurysm of the carotid artery; the purpose of this paper is to present and analyze twelve patients with this syndrome in whom the cause was found to be other than aneurysm. Case reports include three cases due to diabetes, three to paracitoid tumor, one to leukemia and two to ophthalmoplegic migraine.

Aids for differentiating the nonaneurysmal entity from the aneurysmal are listed.

10-3

Tour, Robert L.: *Disturbances of ocular motility in relation to supranuclear lesions*, Am. J. Ophth. 42:873-883 (Dec.) 1956.

This paper describes the most common types of disorders of ocular motility related to lesions in the various portions of the supranuclear pathways.

For convenience the author has discussed the subject under four headings involving four separate, although related, types of control of ocular motility. "The mechanisms involved are:

"1. The frontal mechanism, which is concerned with the control of voluntary conjugate movements.

"2. The disjunctive mechanism which controls convergence and divergence. This is thought to be influenced by both the frontal and occipital mechanisms.

"3. The occipital mechanism, which controls involuntary reflex movements. Examples are the following and fixation reflexes.

"4. The extrapyramidal mechanism, consisting of the vestibular system and the striatal system. These control respectively the righting reflex and the tonus and coordination of the extraocular muscles."

The anatomy of the pathways involved in each of the above mechanisms is discussed briefly. The lesions found in each of the four mechanisms are discussed in more detail.

11-1

Boeder, Paul: *An analysis of the general type of uniocular rotations*, A.M.A. Arch. Ophth., 57:200-206 (Feb.) 1957.

The object of this paper is to demonstrate that it is possible to gain an insight into the mechanics of all types of oculorotations without an elaborate mathematical machinery.

A brief review of the fundamentals of oculorotations is given. Listing's law is stated and discussed. "Listing's law excludes all cyclorotations with respect to the primary position, pure as well as induced ones, and so the general problem regarding the movements of the single eye is to find those rotations that are free of cyclorotation with respect to the primary position. This thorny mathematical problem in spherical kinematics becomes surprisingly simple if we seek the solution via a plane map of the globe." The author has, by means of plotting points on a map of the globe, demonstrated that the eye changes fixation only along a circle through the occipital point. "This restriction—this reduction of the degrees of freedom of oculorotation from 3 to 2—has the remarkable advantage of insuring, for every fixation of the globe, a reproducible orientation, free of cyclorotations with respect to the primary position, no matter from which position and on which path the fixation is reached."

11-2

Breinin, Goodwin M.: *Electromyography—a tool in ocular and neurologic diagnosis*, A.M.A. Arch. Ophth. 57:165-175 (Feb.) 1957.

Application of electromyography (EMG) is discussed in (1) neurogenic palsies of extraocular muscles, (2) progressive nuclear ophthalmoplegia, and (3) nystagmus. Case reports accompany each discussion.

In neurogenic palsies of extraocular muscles the EMG pattern of mild palsies is indistinguishable from normal. Paresis of moderate to

severe degree is indicated by irregular or sparse recruitment, poorly sustained discharge and loss of the "interference" pattern characteristically seen on effort, the action of a muscle, and denervation fibrillations. The motor units in a severe paresis of extraocular muscle are usually of decreased amplitude.

Electromyography has proved useful in a number of cases of pseudoparesis by establishing that the apparently paretic muscle had a normal innervational pattern. Primary and secondary deviations have been determined electromyographically.

Electromyography was performed in two cases of Duane's syndrome. In one the classical explanation for the retraction syndrome was confirmed. In the other the results suggested but did not prove that the basic disturbance was a neurogenic lesion.

In aberrant third nerve regeneration the studies of Bender and Fulton which indicate that misdirection of the regenerating nerve fibers could explain the clinical findings were confirmed in several cases, but the author feels further consideration is required.

In progressive nuclear ophthalmoplegia the observations to date with electromyography suggest that the disease is innervationally more closely allied to muscle pathology than to neurological pathology.

The complete analysis of nystagmus involves (a) the innervation and (b) the response. With electromyography it is possible to record the two simultaneously.

11-3

Breinin, Goodwin M.: *Electromyographic evidence for ocular muscle proprioception in man*, A.M.A. Arch. Ophth., 56:176-180 (Feb.) 1957.

It has now been generally denied that a knowledge of the position of the eyes due to messages from the ocular muscles exists. "The information derived from vision and the innervational urge are said to be adequate to explain our awareness or lack of awareness of eye position." However, Dr. Breinin feels that the theory that extraocular muscles lack a mechanism for recording muscle tension would place them so far apart from other skeletal muscle as to arouse wonder. The extraocular muscles do have anatomic and pharmacologic peculiarities.

Observations have been made with electromyography which indicate that a mechanism does exist for signaling changes in muscle tension. These observations are discussed. It is not implied, however, that this mechanism provides muscle position sense or awareness.

11-4

Breinin, Goodwin M.: *Quantitation of extraocular muscle innervation*, A.M.A. Arch. Ophth., 57:644-650 (May) 1957.

Dr. Breinin has made another important contribution to the field of electrophysiology by developing an instrument for recording extraocular innervations which will greatly facilitate the analysis of this procedure. In this instrument (made up of a full wave integrator and differentiator), integration of the energy developed in specific time intervals is proportioned to the discharge intervals. This discharge is fed into a second beam of the dual oscilloscope or can be written out on an ink writer.

Eight electromyograms clarifying the innervations in the various positions of gaze further clarify the level of innervation of the extraocular muscles. The effect of a lens or prism in augmenting or inhibiting accommodative vergences can also be evaluated. With this instrument it will be possible to compare a lens-induced vergence innervation and that due to a linear distance of an accommodative symbol. Studies evaluating such changes in accommodative strabismus are now being done, as are also quantitative analyses of the action of drugs upon extraocular muscle innervation, particularly in studies of myasthenia gravis.

13-1

Naylor, E. J.; Shannon, T. E., and Stanworth, A.: *Stereopsis and depth perception after treatment for convergent squint*, Brit. J. Ophth., 40:641-651 (Nov.) 1956.

This survey arose from a paper reporting the improvement of binocular vision after operations for squint in patients in whom restoration of the binocular vision was not expected.

Eighteen per cent of a group of patients who had been discharged as nonbinocular had improved in binocular vision in three years; this seemed to prove that binocular functions will improve with the passage of time providing the visual axes are rendered approximately parallel. An early operation is stressed, although other cases in which operation was postponed are discussed.

In this series an analysis was done of the results in 151 patients who were without fusion before operation, and the series also extended to include 112 patients with fusion before operation and 20 patients discharged as cured from the orthoptic department without operation. The value of the stereopsis was deter-

mined by depth perception tests on the synoptophore, the cover test, and the amplitude of fusion found on the synoptophore. Patients with a large fusion amplitude and a satisfactory cover test had better depth perception.

Five questions are discussed: (1) the factors determining the presence of stereopsis after operation, (2) the degree of depth perception that is present in patients who show stereopsis on the synoptophore, (3) the factors determining the presence of good depth perception in patients with postoperative stereopsis, (4) the importance of clinical tests as a guide to the degree of depth perception present, (5) the value of binocular vision in patients who show stereopsis on the synoptophore, but have subnormal depth perception.

In summary, the authors state that 263 patients were examined postoperatively, and depth perception tests were carried out on 60 patients who had stereopsis, 12 patients without stereopsis, and 20 patients who had been discharged from the orthoptic department without surgical treatment.

13-2

Ogle, Kenneth N., and Groch, Judith: *Stereopsis and unequal luminosities of the images in the two eyes*, A.M.A. Arch. Ophth., 56:878-895 (Dec.) 1956.

The authors have presented their study concerning the extent to which a difference in the luminosities of the images in the two eyes can affect binocular vision and depth perception. The conclusion drawn is "that decreasing the luminosities of the image in one eye does not alter the physiologic disparity between the images of the two eyes or the stereoscopic depth."

14-1

Schlossman, Abraham: *Correlation between preoperative measurements and postoperative results in horizontal concomitant strabismus*, EENT Monthly, 36:115 (Feb.) 1957.

Every surgical procedure must be carefully planned and all the data that can be obtained from history and from objective and subjective examinations should be taken into consideration. Results are often unpredictable and a certain amount of skepticism prevails among the most experienced surgeons regarding the advisability of giving definite rules to guide the ophthalmologist in planning his procedure.

Some differences in surgical results are:

"1. Equal amounts of surgical correction often give different results in different patients.

"2. Different types and amounts of surgical correction may give identical results in cases with similar measurements.

"There are three factors which favor good surgical results: (1) The same amount of surgery usually yields a greater amount of correction in patients with a higher degree of strabismus than it does in patients with a lesser degree of squint. (2) If glasses correct at least one half or more of the esotropia, then any amount or type of surgery which is reasonably correct for the esotropia will give excellent results. This is probably due to the fact that such patients have fairly good fusional ability. (3) Finally, in hereditary cases, if the ophthalmologist can use information gleaned from the end result of surgery in one member of the family in which similar types of squint prevail, it is probably the safest guide in planning surgical correction on another member of the same family."

14-2

Simpson, Derek G.: *Marcus Gunn phenomenon following squint and ptosis surgery*, A.M.A. Arch. Ophth., 56:743-748 (Nov.) 1956.

The typical clinical picture of the Marcus Gunn phenomenon presents a unilateral congenital ptosis in which there is a retraction of the lid on opening the jaw. Variations which may occur in the typical picture are listed.

The following etiologic hypotheses are discussed: (1) abnormal nervous connections, (2) functional interference and (3) atavistic reversion.

A case report is given of a 28-year-old woman with a left ptosis present since birth associated with a down and out deviation of the left eye. No jaw winking had been noted at any time. Surgical treatment was done in three stages: left resection of internal rectus and tenotomy of the external rectus, resection of the superior rectus and recession of the inferior rectus, and shortening of the levator tendon by the Blaskowicz type of operation. The cosmetic result of the ptosis and squint was good but an obvious lid retraction was noted when the jaw was opened quickly.

The etiologic hypotheses of the Marcus Gunn phenomenon are discussed in an attempt to explain the above case.

16-1

Alpern, Mathew, and Wolter, J. Reimer: *The relation of horizontal sac-*

cadic and vergence movements, A.M.A. Arch. Ophth., 56:685-690 (Nov.) 1956.

"When an observer with normal eyes is instructed to shift his gaze suddenly from one fixated point in the visual field to a second, the time characteristics of the movements of the eyes depend upon the positions of the two points within the field. Dodge pointed out that if the two points are confined to the horizontal plane which includes the centers of rotation of the two eyes, then two basic types of movements can be differentiated according to the velocity of the movement:

"(a) If the two points are confined approximately to an objective frontoparallel plane, then the movement is very rapid. The velocity of this movement may be higher than 400 degrees per second and increases as the separation of the fixated points increases. Such movements are called saccadic movements.

"(b) If the points are so arranged that they fall on a forward extension of the median body plane, then the movement is much slower. . . . For our observer the average velocity is approximately 8 degrees per second, and Dodge reported values ranging from 25 degrees per second to 10 degrees per second. Such movements are called vergence movements.

"Dodge also pointed out that if the points (still in the horizontal plane) are intermediate between these two positions then the change of fixation of the eyes from one point to the second will contain both of the basic types of movements. Thus, if the near object is shifted so that it falls along the line of sight of the right eye (while it fixates the far object) then the result of binocular shift from far to near contains a saccadic fast component to the right and a slow component of convergence. . . . It is interesting to point out that for this observer both eyes move. This confirms Dodge's results completely although such findings have been denied by Bielschowsky, Scobee and Breinin."

The explanation of the difference in velocity of these two types of movements is discussed. One explanation is "that in all convergence movements of the eyes, each eye is retarded more or less by the tendency to move sympathetically in the same direction of the other eye." Another explanation may be found in the morphological study of the extraocular muscles. "The fibers of the extraocular muscles are supplied by two different types of nerve fibers, both of which apparently are efferent:

"(a) Coarse, medullated nerve fibers form typical motor end-plates . . . which innervate the large muscle fibers only.

"(b) Small nonmedullated nerve fibers form either loop-like endings close to the motor end-plates or delicate network-like endings on the muscle fibers." Innervation from these nerves may be either to the large muscle fibers or to the small specific muscle fibers.

"The final common paths of saccadic movements are formed by the thick somatic nerve fibers, while the final common paths for vergence movements are formed by this autonomic fibers. . . . Finally this speculation provides a basis for the understanding of the ability of an eye muscle to carry out a yoked movement together with a muscle of the other eye at one moment and at another moment during the same change of fixation carry out a second yoked movement with the latter muscle's antagonist."

16-2

Ogle, Kenneth N., and Martens, Theodore G.: *On the accommodative convergence and the proximal convergence*, A.M.A. Arch. Ophth., 57:702-715 (May) 1957.

Some interesting investigations have been conducted by Drs. Ogle and Martens regarding accommodative-convergence synkinesis and proximal influence in the total convergence which the eyes would require for a given viewing distance. Information of this nature would be pertinent in writing refractive corrections, better understanding of asthenopia resulting from heterophoria, as well as the more general problem of strabismus. Conclusions have previously been made by Asher and Guzzinati regarding a person's conscious estimate of the position of an object as related to accommodation-convergence. It was mentioned as a factor in asthenopia, but in particular it was found to affect convergence to the extent that convergence insufficiency could be reduced nearly in half.

The two types of techniques used for this testing were: (1) the heterophoria method and (2) the fixation-disparity method. Results of these tests are summarized in graphs and the testing equipment is clarified by drawings.

1. The heterophoria method used two common techniques: the Maddox rod-light fixation combined with a test object; the change stimulus in accommodation being induced by plus or minus lenses. The ratio $(A-C)/A$ was formulated for this test. Subjects for this test were 28 resident physicians and technicians from the Mayo Clinic between the ages of 19 and 39. They all had normal vision without a correction—the test was done without a

correction. A geometric relationship of the visual angle was recorded, making it possible to compute and compare the actual change in the fusion-free convergence and the change when a stimulus was inserted.

2. The fixation-disparity method rests on the fact that "if a muscular imbalance exists, even in normal binocular observation, the eyes will fail to converge accurately at the point of fixation by a small angle, overconverging in esophoria and underconverging in exophoria." For this test there were 104 subjects (for the most part patients in the section of Ophthalmology) ranging in age from 14 to 72. Of these there were patients with myopia and hyperopia; others had esophoria for near and some had exophoria for near. Results showed no significant relationship existing between refractive errors, but both the accommodative and proximal convergence were reduced in convergence insufficiency and the accommodative convergence was increased in convergence excess.

Summaries from the results of each of these methods have been tabulated with their comparative statistics being given on a third table. Data of two representative cases, one of an overconvergence at near and the other of a large exophoria for near (with presbyopia), are also presented.

In evaluating the techniques, the authors were inclined to favor the fixation-disparity method because it gave a more stabilized type of data, since this test requires fusion and both eyes are subject to the same stimulus to accommodation. As a group, the patients with myopia had a higher (A-C)/A ratio than patients with hyperopia. This was interpreted to mean that the innervation necessary to cause a given change in accommodation of a patient with myopia was greater than that of a patient with hyperopia or a normal subject. Convergence insufficiency was associated both with a decreased (A-C)/A ratio and with a decreased proximal convergence.

16-3

Passmore, Jack W., and MacLean, Florence: *Convergence insufficiency and its management*, Am. J. Ophth., 43: 448-456 (March) 1957.

This paper presents a discussion and evaluation of the orthoptic treatment of 100 patients with convergence insufficiency. This condition was found most often in the young adult age group. The etiology was usually not ascertained. Symptoms were the usual headaches and discomfort and blurring for near work. Symptoms occurred most commonly late in the day.

The objective findings are usually a remote (beyond 9.5 cm.) near point of convergence (NPC), although this may be normal. Measurements with the Maddox rod are not diagnostic. Convergence on a near test light with a red filter before one eye usually revealed a difference of about 35 mm. from that without the red filter. Prism convergence is poor at near and there may be suppression through the range of fusion. Many of the patients had poor superimposition on the major amblyoscope.

Treatment was by means of orthoptics and and it was noted that no operation or prismatic correction in the glasses was used or indicated. The following orthoptic exercises are described in detail: pin-to-nose, separator and pin exercise, pencil-to-pencil, loose prism convergence for distance and near, amblyoscope exercises, photoscope, split stereograms, orthofusor and stereoscope exercises. Suggestions are made regarding the stage in treatment at which each of these exercises is most helpful. Goals are listed which should be reached with each exercise if symptoms are to be prevented from recurring.

Orthoptic treatment both at home and in the orthoptic clinic results in a high percentage of cures. Prismatic correction and surgical treatment are rarely necessary and are inadvisable.

17-1

Folk, Eugene R.: *Superior oblique tendon sheath syndrome*, A.M.A. Arch. Ophth., 57:39-40 (Jan.) 1957.

A case is presented which demonstrates all the characteristics of the superior oblique tendon sheath syndrome. The characteristics of this syndrome are paralysis of the inferior oblique, little or no overaction of the superior oblique, depression of the eye on adduction and limitation to passive elevation of the adducted eye. Brown felt that this syndrome is due to a congenitally short sheath of the superior oblique.

The author's patient was treated surgically on two occasions. At the time of the first procedure the superior oblique was isolated and its sheath stripped almost to the trochlea. Afterward an esotropia and left hypotropia still remained. A second operation showed that the sheath had re-formed. The sheath of the superior oblique was again stripped and a strengthening procedure performed on the inferior oblique. Much better results were obtained at this time.

This case demonstrates that stripping of the superior oblique sheath alone is of little value and that probably only a strengthening procedure on the inferior oblique would have had no result.

17-2

Friedman, Joseph Jay: *Adhesion of the superior oblique muscle fascial sheath*, Am. J. Ophth., 43:115-118 (Jan.) 1957.

A review of the literature is given regarding cases of muscular and fascial anomalies of the superior oblique. In only one instance was any mention made of a case of adherence of the superior oblique muscle sheath to the sheath of the medial rectus muscle. The authors report such a case: a child, age 2, who showed a restriction of action of the superior oblique because of adhesion of its sheath to the insertion of the medial rectus. There was a compensatory overaction of the opposing inferior oblique. The patient preferred to keep her head down with chin on chest when looking straight ahead.

Surgical operation was performed with freeing of the adhesions of the superior oblique sheath as well as recession of the medial rectus (4.0 mm.). Marked cosmetic improvement resulted, and the patient now holds her head in a normal position. It is too soon to tell whether or not a functional cure will be obtained.

17-3

Lebensohn, James E.: *Pupillary hypophoria and hyperphoria*, Am. J. Ophth., 42:770-771 (Nov.) 1956.

In the presence of bilateral aphakia a disparity in the pupillary levels occasions a hyperphoria or hypophoria that is present without, but not with, the corrective lenses. Two case reports are presented to illustrate this point.

18-1

Allen, Henry F.: *A new picture series for preschool vision testing*, Am. J. Ophth., 44:38-41 (July) 1957.

Comprehensive testing of preschool children must be done in order to recognize and prevent amblyopia. The E and Sjogren hand offer the best methods. Some children, however, do not understand or have any interest in them. It is for these children that a new type of chart with pictures has been devised. Most three-year-old children work best at fifteen feet.

Pictures should be chosen that have an association for the child, e.g., birthday cake, flowers, telephone, etc. The pictures that are chosen must be made to co-relate to known levels of visual acuity. Illumination must be controlled.

The new type of test has a series of pictures incorporated into a small ring binder—one to a side. They correspond with a thirty foot E chart. "Differences of even one line in Snellen acuity are reflected in corresponding shortening of recognition distance."

This test is to complement the known type of testing used.

18-2

Prince, Jack H.: *A new test for visual malingering*, Am. J. Ophth., 42:776-777 (Nov.) 1956.

Two charts are described that have been designed to detect malingering. One chart consists of letters in which the differences between their visual thresholds have been used to produce a situation in which the last letter of any line has the same value as the first letter on the next line following it. The subject who reads all of a line, but claims he is unable to read any of the next line, is suspect.

The second chart consists of Landolt rings in which the size is not related to the width of the break. The width of the break is the criterion for assessing visual acuity, whereas the subject assumes it is the over-all size.

The test should always be applied with full correction to avoid any astigmatic influence on the Landolt rings.

1958

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Chattanooga, Tenn.
Sponsor: Dr. W. H. Steel, Jr.
- Brandt, Miss Idella; 1945
120 Grand St.
White Plains, N. Y.
Sponsor: Dr. Christopher Wood
- Broadbent, Mrs. Jacqueline Owens; 1945
2845 Van Aken Blvd.
Shaker Heights, Ohio
Sponsor: Dr. Webb P. Chamberlain, Jr.
- Brown, Mrs. Elizabeth Coltart; 1940
49½ W. Second St.
Mansfield, Ohio
Sponsor: Dr. W. Max Brown
- Brown, Mrs. I. G. L.; 1955
100-103 Medical Dental Bldg.
12th Avenue and Second Street, W.
Calgary, Alta., Canada
Sponsors: Dr. J. A. McLean
Dr. Ernest A. Johnson
- Brunn, Miss Agatha; 1939
111 Glendale Rd.
Upper Darby, Pa.
Sponsor: Dr. Patrick J. Kennedy
- Bullwinkel, Mrs. Lola L.; 1953
202 Dover St.
Easton, Md.
Sponsor: Dr. H. G. Bullwinkel
- Burns, Mrs. Mary Smith; 1951
6850 N. E. Killingsworth
Portland, Ore.
- Burri, Miss Clara; 1939
205 W. Lullwood
San Antonio, Texas
Sponsor: Dr. W. A. Reily
- Butler, Mrs. Leta Counihan; 1950
2675 Henry Hudson Pkwy.
Riverdale, N. Y.
- Butts, Mrs. Margaret E.; 1946
4088 Jenkins Arcade
Pittsburgh 22, Pa.
Sponsor: Dr. Raymond J. Gray
- Campbell, Mrs. Mary Thomas; 1953
1904 Indian Head Rd.
Ruxton 4, Md.
- Capobianco, Miss Nancy; 1949
Policlinica
Clinica Oculistica dell Universita di Roma
Rome, Italy
Sponsor: Dr. G. B. Bietti
- Carbajal, Mrs. Jovita C.; 1951
1430½ Michigan Ave.,
Los Angeles 33, Calif.
- Cassin, Mrs. Barbara; 1951
c/o Dr. Cassin
Department of Physiology
College of Medicine
University of Florida
Gainesville, Fla.
- Casten, Mrs. Barbara Hubbard; 1947
120 Bonita Ave.
Piedmont, Calif.
- Chalat, Mrs. Joanne S.; 1950
212 David Whitney Bldg.
Detroit 26, Mich.
- Chan, Mrs. Doris H.; 1955
White Memorial Hospital
1720 Brooklyn Ave.
Los Angeles 33, Calif.
Sponsor: Dr. George K. Kambara

- Chesner, Mrs. Merelyn Schroeder; 1949
Milwaukee Ophthalmic Institute
720 N. Jefferson St.
Milwaukee 2, Wis.
Sponsor: Dr. John B. Hitz
- Cibis, Mrs. Lisa; 1956
Department of Ophthalmology
Washington University School of Medicine
640 S. Kingshighway Blvd.
St. Louis 10, Mo.
Sponsor: Dr. Bernard Becker
- Clark, Mrs. Ann Fox; 1953
Box 0102
Mount Hermon, Mass.
- Clausen, Miss Georgia; 1946
1826 State St.
Santa Barbara, Calif.
Sponsor: Dr. Michel Loutfallah
- Clavell, Miss Joan; 1951
Orthoptic Clinic
General Hospital
Calgary, Alta., Canada
Sponsor: Dr. L. C. Cody
- Coelis, Miss Susan A.; 1955
Grady Memorial Hospital
80 Butler St., S. W.
Atlanta 3, Ga.
Sponsor: Dr. J. P. Deweer
- Connelly, Miss Grace; 1954
Ophthalmic Laboratory
14540 Hamlin St.
Van Nuys, Calif.
Sponsor: Dr. Mayo J. Poppen
- Cooper, Mrs. Grace Blunt; 1947
10954 O'Dell Ave.
Sunland, Calif.
- Cooper, Miss Loraine H.; 1948
208 Clayton Medical Bldg.
35 N. Central Ave.
Clayton 5, Mo.
Sponsor: Dr. F. W. Luedde
- Cory, Mrs. Elizabeth Christ; 1953
602 N. W. 23rd Pl.
Miami 35, Fla.
- Crane, Mrs. Shirley; 1949
859 Monte Verde Dr.
Arcadia, Calif.
- Cronin, Miss Mary L.; 1948
Section of Ophthalmology
Mayo Clinic
Rochester, Minn.
Sponsor: Dr. T. G. Martens
- Crowe, Mrs. Mary Thompson; 1951
313 Calle Miramar
Redondo Beach, Calif.
- Crush, Miss Margaret; 1947
Orthoptic Center
4 W. Fourth St.
Cincinnati 2, Ohio
Sponsor: Dr. Donald J. Lyle
- Daetwyler, Mrs. Marilyn Marqua; 1952
4817 E. 13th
Tulsa, Okla.
- Darr, Mrs. Lillian; 1939
4539 Central Ave.
Western Springs, Ill.
- Davidson, Miss Joanne L.; 1954
Metropolitan Health Unit No. 3
1530 W. Eighth Ave.
Vancouver 9, B. C., Canada
Sponsor: Dr. John A. McLean
- Davis, Miss Bobbie; 1956
Suite 12
2708 St. John's Ave.
Jacksonville, Fla.
Sponsor: Dr. W. J. Knauer, Jr.
- Davis, Miss Diane; 1956
Suite 30
3195 Granville St.
Vancouver, B. C., Canada
Sponsor: Dr. J. F. Minnes
- Dean, Mrs. Alice McPhail; 1942
7234 Lakewood Blvd.
Dallas, Texas
- Deeley, Miss Mary R.; 1948
313 S. 17th St.
Philadelphia 3, Pa.
Sponsor: Dr. Francis Heed Adler
- De Gowin, Mrs. Karen; 1957
950 E. 59th St.
Chicago 37, Ill.
Sponsor: Dr. Frank W. Newell
- DeMars, Miss Eva M.; 1940
2601 Parkway
Philadelphia 3, Pa.
Sponsor: Dr. William E. Krewson, III

Dendy, Miss Hazel; 1956
Orthoptic Department, O.P.D.
Bellevue Hospital
First Avenue and 26th Street
New York, N. Y.
Sponsor: Dr. Goodwin M. Breinin

Denger, Mrs. Lucille; 1956
208 Clayton Medical Bldg.
35 N. Central
Clayton 5, Mo.
Sponsor: Dr. F. W. Luedde

Dewing, Mrs. Rosalie Nestor; 1946
Box 79
Route 1
McLean, Va.

Dobrasz, Miss Frances; 1956
Buffalo Orthoptic Center
52 Maple St.
Buffalo 4, N. Y.
Sponsor: Dr. Howard H. Higgs

Donahue, Miss Mary E.; 1951
Hartford Lions Club Orthoptic Clinic
Hartford Hospital
Seymour Street
Hartford, Conn.
Sponsor: Dr. Arthur C. Unsworth

Drye, Miss Laura B.; 1941
Orthoptic Clinic
Eye and Ear Hospital
230 Lothrop St.
Pittsburgh 13, Pa.
Sponsor: Dr. Murray F. McCaslin

DuBlon, Miss Janet; 1950
14 Forest Ave.
North Baldwin, N. Y.
Sponsor: Dr. Milton T. Gailard

Dunnam, Mrs. Betty Storer; 1947
Room 109
414 N. Camden Dr.
Beverly Hills, Calif.
Sponsor: Dr. James R. Cogan

Durham, Mrs. Harriet F.; 1948
Professional Bldg.
Augustine Cut-off
Wilmington, Del.
Sponsor: Dr. Davis G. Durham

Eaves, Mr. Bert O.; 1939
366 Mt. Prospect Ave.
Newark, N. J.
Sponsor: Dr. Berta D. Rados

Ede, Mrs. Mabel L.; 1955
Eye Clinic
University of California Medical Center
Parnassus and Third
San Francisco 22, Calif.
Sponsor: Dr. George S. Campion

Eisenmann, Mrs. Olive Boyle; 1941
Hull-Dobbs Ranch
Walls, Miss.

Engel, Miss Dolores; 1945
1684 N. Prospect Ave.
Milwaukee 2, Wis.
Sponsor: Dr. Raymond C. Warner

Erkfitz, Miss Anne; 1954
1633 David Whitney Bldg.
Detroit 26, Mich.
Sponsor: Dr. A. D. Ruedemann

Eustis, Miss Ann T.; 1951
Ocular Motility Clinic
Washington Hospital Center
100 Irving Center
Washington 1, D. C.
Sponsor: Dr. Arlington C. Krause

Evans, Mrs. Mildred Smith; 1940
14 W. Mt. Vernon Pl.
Baltimore 1, Md.
Sponsor: Dr. Frank B. Walsh

Eyles, Miss Marianne A. D.; 1946
4635 White Oak Ave.
Encino, Calif.
Sponsor: Dr. S. Rodman Irvine

Ferguson, Miss Constance M.; 1955
Birmingham and Midland Counties
Eye Hospital
Birmingham, England

Ferney, Miss Sally J.; 1955
1652 David Whitney Bldg.
Detroit 26, Mich.
Sponsor: Dr. A. D. Ruedemann

Fike, Miss Eleanor E.; 1955
Eye, Ear, Nose and Throat Clinic
Box 367
Moultrie, Ga.
Sponsor: Dr. James R. Paulk

Fisher, Miss Ruth; 1947
924 Republic Bldg.
Denver 2, Colo.
Sponsor: Dr. Donald H. O'Rourke

- Fitton, Miss Margaret; 1956
Department of Ophthalmology
New York State University
Brooklyn, N. Y.
Sponsor: Dr. Richard Troutman
- Fonzi, Miss Helen C.; 1956
225 East Third St.
Williamsburg, Pa.
- Fowler, Miss Frances; 1946
4635 White Oak Ave.
Encino, Calif.
Sponsor: Dr. S. Rodman Irvine
- Franklin, Mrs. Frances; 1953
Orthoptic Clinic
Indiana University Medical Center
Indianapolis 7, Ind.
Sponsor: Dr. Fred M. Wilson
- Gjullin, Mrs. Helen Innes; 1954
P. O. Box 405
Corvallis, Ore.
Sponsor: Dr. George P. Lyman
- Glaser, Miss Norma; 1952
400 E. 80th St.
New York 21, N. Y.
Sponsor: Dr. Bernard Fread
- Goggin, Mrs. Elizabeth A.; 1947
325 Doctors Bldg.
Minneapolis, Minn.
Sponsor: Dr. Malcolm A. McCannel
- Goldberg, Miss Evelyn L.; 1951
111-32 76th Ave.
Forest Hills, N. Y.
Sponsor: Dr. Edward L. Seretan
- Goldthwait, Miss Lucy F.; 1950
Lions Orthoptic Clinic
33 Pearl St.
Springfield 3, Mass.
Sponsor: Dr. William F. Donoghue
- Gonzalez, Miss Maria; 1943
116 S. Michigan Ave.
Chicago 3, Ill.
Sponsor: Dr. Theodore M. Shapira
- Gould, Mrs. Audrey; 1955
Orthoptic Clinic
Middlesex General Hospital
New Brunswick, N. J.
Sponsor: Dr. William Rubin
- Greer, Mrs. Ellen Watson; 1950
1111 Arbor
Houston 4, Texas
Sponsor: Dr. Louis J. Girard
- Greer, Mrs. Joan Fehrman; 1955
611 Adams St.
Rapid City, S. D.
Sponsor: Dr. E. S. Palmerton
- Groves, Miss Patricia B.; 1946
Department of Ophthalmology
Cleveland Clinic
2020 E. 93rd St.
Cleveland 6, Ohio
Sponsor: Dr. Roscoe J. Kennedy
- Gunter, Mrs. Vernelle Boyd; 1952
161 W. Cheves St.
Florence, S. C.
Sponsor: Dr. J. Howard Stokes
- Gutheim, Miss Inge; 1949
17329 Monica
Detroit 21, Mich.
Sponsor: Dr. H. Saul Sugar
- Halas, Mrs. Marilyn Evans; 1950
Department of Ophthalmology
Cleveland Clinic
2020 E. 93rd St.
Cleveland 6, Ohio
Sponsor: Dr. Roscoe J. Kennedy
- Hall, Mrs. Geneva Fallon; 1950
805 26th St.
Sacramento, Calif.
Sponsor: Dr. Theodore C. Zeman
- Hall, Mrs. Jane M.; 1950
704 Congress St.
Portland 4, Maine
Sponsor: Dr. Richard J. Goduti
- Hamilton, Mrs. Catherine M.; 1956
Suite 421
604 Columbia St.
New Westminster, B. C., Canada
Sponsor: Dr. H. S. Hamilton
- Hamilton, Mr. Robert E.; 1956
Ophthalmic Laboratory
812 Pine Ave.
Long Beach, Calif.
Sponsor: Dr. Robert G. Thornburgh
- Hardy, Mrs. Elva Florrid; 1949
1446 Ninth Ave.
San Francisco, Calif.
- Harley, Miss Elisabeth R.; 1941
McPherson Hospital
1110 W. Main St.
Durham, N. C.
Sponsor: Dr. S. D. McPherson, Jr.

- Hathel, Miss Doris Jean; 1957
108 Beaumont Medical Bldg.
3720 Washington Blvd.
St. Louis 8, Mo.
- Healy, Miss Electra; 1941
4753 Broadway
Chicago 40, Ill.
Sponsor: Dr. William J. Noonan
- Heath, Mrs. Joyce McEvers; 1949
V Bar B Ranch
Issaquah, Wash.
- Hedden, Mrs. Dorothy Parkhill; 1949
Chicago Orthoptic Institute
203 N. Wabash Ave.
Chicago 1, Ill.
Sponsor: Dr. Warren F. Smith
- Heffernan, Miss Mary E.; 1956
Truesdale Hospital
218 Calvin St.
Fall River, Mass.
Sponsor: Dr. John W. Henderson
- Heinen, Miss Lydia K.; 1939
Motility Clinic
Illinois Eye and Ear Infirmary
904 W. Adams St.
Chicago, Ill.
Sponsor: Dr. William F. Hughes, Jr.
- Hellebo, Miss Helen; 1947
400 Medical Arts Bldg.
Minneapolis 2, Minn.
Sponsor: Dr. Walter M. Fink
- Hempstead, Miss Mary; 1947
Gailey Eye Clinic
1008 N. Main St.
Bloomington, Ill.
Sponsor: Dr. Watson Gailey
- Henderson, Mrs. Dorothy D.; 1950
Orthoptic Clinic
Truesdale Hospital
218 Calvin St.
Fall River, Mass.
Sponsor: Dr. Paul P. Dunn
- Henderson, Miss Margaret M.; 1954
Suite 201
2525 Pine St.
Vancouver, B. C., Canada
Sponsor: Dr. C. E. Davies
- Hermes, Miss Carol; 1957
24 Park Dr.
Dayton 10, Ohio
Sponsor: Dr. Malachi W. Sloan, II
- Herslof, Mrs. Serena; 1941
3444 N. Hackett Ave.
Milwaukee 11, Wis.
Sponsor: Dr. W. H. Bennett
- Holoviak, Miss Helen; 1940
128 MacDougal St.
New York 12, N. Y.
Sponsor: Dr. Sidney A. Fox
- Hornlein, Miss Jean; 1954
Grace New Haven Hospital
789 Howard Ave.
New Haven 4, Conn.
Sponsor: Dr. Clement C. Clarke
- Hughes, Mrs. Ann; 1952
537 Woodland Ave.
Mountainside, N. J.
Sponsor: Dr. Richard Baruch
- Hughes, Miss Marguerite D.; 1956
150 E. Broad St.
Columbus 15, Ohio
Sponsor: Dr. Jacob Moses
- Hunt, Miss Marlene; 1955
Orthoptic Clinic
50 Medical Arts Bldg.
Victoria, B. C., Canada
Sponsor: Dr. J. T. Cruise
- Hurt, Miss Jane; 1956
Department of Ophthalmology
Washington University School of Medicine
640 Kingshighway Blvd.
St. Louis 10, Mo.
Sponsor: Dr. Bernard Becker
- Iacobucci, Miss Ida; 1957
Department of Ophthalmology
University Hospital
Outpatient Building
Ann Arbor, Mich.
Sponsor: Dr. John W. Henderson
- Jacino, Miss Julia A.; 1956
Spokane Eye Clinic
S. 427 Bernard
Spokane, Wash.
Sponsor: Dr. C. L. Gates
- Jackson, Miss F. Elizabeth; 1945
Orthoptic Clinic
St. Vincent's Hospital
Erie, Pa.
Sponsor: Dr. James H. Delaney

- Jaffee, Mrs. Fern Block; 1946
1370 Arbor Ave.
Highland Park, Ill.
- James, Miss Jillian A.; 1954
Orthoptic Centre
595 E. Colorado
Pasadena, Calif.
Sponsor: Dr. George E. Morgan
- Janzing, Mrs. Dorothy Burton; 1948
10613 S. E. 248th
Kent, Wash.
Sponsor: Dr. Bruce McClellan
- Jaquith, Mrs. Pearl E.; 1953
Box 511
Brawley, Calif.
Sponsor: Dr. George Jaquith
- Jones, Mrs. Eloise Allen; 1955
248 Plaza Theatre Bldg.
Kansas City, Mo.
Sponsor: Dr. John McLeod
- Kennelly, Mrs. Miriam Englert; 1949
288 Woodruff Dr.
Webster, N. Y.
- Kiehnoff, Mrs. Emily Schoech; 1944
5811 Fredrick
Omaha, Neb.
- Kirkland, Mrs. Ruth Morris; 1948
2000 Seventh St.
Tuscaloosa, Ala.
Sponsor: Dr. T. N. Kirkland, Jr.
- Knauber, Miss Edna; 1939
Orthoptic Clinic
Manhattan Eye, Ear and Throat Hospital
210 E. 64th St.
New York 21, N. Y.
Sponsor: Dr. David H. Webster
- Knight, Mrs. Geraldine Wood; 1949
332 Cobb Bldg.
Seattle 1, Wash.
Sponsor: Dr. R. F. Kaiser
- Koehler, Miss Virginia D.; 1949
Department of Ophthalmology
Reading Hospital
West Reading, Pa.
Sponsor: Dr. John M. Wotring
- Kramer, Mrs. Louisa Wells; 1939
1779 Massachusetts Ave., N. W.
Washington 6, D. C.
Sponsor: Dr. Ronald A. Cox
- Kramer, Miss Mary E.; 1941
Room 26
Park Central Medical Bldg.
550 W. Thomas Rd.
Phoenix, Ariz.
Sponsor: Dr. Paul H. Case
- Kratka, Mrs. Zelda; 1953
504 Medical Arts Bldg.
Wilmington, Del.
Sponsor: Dr. William H. Kratka
- Kreska, Miss Anna V. M.; 1955
1034 Spring St.
Reading, Pa.
Sponsor: Dr. Harold L. Strause
- Kubek, Miss Jean; 1941
2025 Blais Ave.
Daytona Beach, Fla.
- Kuecken, Mrs. Shirley Loud; 1946
8715 Monsanto
Cincinnati 31, Ohio
- Kuhn, Mrs. Anita G.; 1950
112 Rimbach St.
Hammond, Ind.
- Kukora, Miss Josephine S.; 1946
Orthoptic Clinic
Department of Ophthalmology
Henry Ford Hospital
Detroit 2, Mich.
Sponsor: Dr. Jack S. Guyton
- Lancaster, Miss Julia; 1939
Sacramento Orthoptic Laboratory
2720 Capitol Ave.
Sacramento, Calif.
Sponsor: Dr. Ulrich A. Fritschi
- Langley, Miss Charlotte; 1953
Dallas Medical and Surgical Center
4105 Live Oak St.
Dallas, Texas
Sponsor: Dr. Ronald M. Burnside
- Lasher, Miss Beverly J.; 1951
1633 David Whitney Bldg.
Detroit 26, Mich.
Sponsor: Dr. A. D. Ruedemann
- Laughlin, Mrs. Elsie; 1941
Department of Ophthalmology
Iowa University Hospitals
Iowa City, Iowa
Sponsor: Dr. Alson E. Braley

Lawton, Mrs. Mary Phillips; 1948
Heckman's Lane
Summit Lawn, Allentown, Pa.

Lewis, Miss Margaret; 1958
517 Third St.
New Westminster, B. C., Canada
Sponsor: Dr. Everett F. Raynor

Linnington, Mrs. Barbara Blair, 1955
744 Foster Rd.
Richmond, B. C., Canada
Sponsor: Dr. John A. McLean

Lister, Miss Marion P.; 1953
Orthoptic Clinic
717 Medical Dental Bldg.
Vancouver 1, B. C., Canada
Sponsor: Dr. John A. McLean

Lorenger, Mrs. Lorraine Lucas; 1950
414 David Whitney Bldg.
Detroit 16, Mich.
Sponsor: Dr. Edmond L. Cooper

Ludwig, Miss Mildred M., 1950
2853 W. Dickens Ave.
Chicago 47, Ill.

Lundean, Mrs. Marguerite S., 1940
Rochester Orthoptic Center
221 Alexander St.
Rochester 7, N. Y.
Sponsor: Dr. John G. Gipner

Lunn, Miss Catherine T.; 1953
Orthoptic Clinic
Health Centre for Children
Vancouver General Hospital
Vancouver 9, B. C., Canada
Sponsor: Dr. John A. McLean

Lutz, Miss Matilda; 1950
Address not known

MacLean, Miss Florence; 1952
Ocular Research Unit
Department of Ophthalmology
Main Eye Clinic
Walter Reed Army Medical Center
Washington, D. C.
Sponsor: Dr. Austin Lowrey, Jr.

Madaire, Miss Dorothea; 1945
Orthoptic Clinic
Southern California Permanente
Medical Group
9985 Sierra Ave.
Fontana, Calif.
Sponsor: Dr. J. F. Kleckner

Maderer, Mrs. Anita Bargmann; 1939
Buffalo Orthoptic Center
52 Maple St.
Buffalo 4, N. Y.
Sponsor: Dr. Howard H. Higgs

Malcolm, Miss Nancy; 1952
717 Medical Dental Bldg.
Vancouver 1, B. C., Canada
Sponsor: Dr. John A. McLean

Mansfield, Mrs. Danele Stevens; 1940
716 Florida Ave.
Oak Ridge, Tenn.

Marsh, Miss Eleanor T.; 1945
101 Braddock St.
Rochester 12, N. Y.
Sponsor: Dr. Charles T. Sullivan

Marshall, Miss Louise; 1947
Address not known

Martz, Mrs. Dolly H.; 1946
608 W. Third St.
Harrisburg, Pa.
Sponsor: Dr. George E. Martz

Masin, Mr. Henry B.; 1955
7460 S. W. 36th St.
Miami, Fla.
Sponsor: Dr. Kenneth S. Whitmer

Matz, Miss Marilyn; 1955
414 David Whitney Bldg.
Detroit 16, Mich.
Sponsor: Dr. Edmond L. Cooper

Maxwell, Mrs. Marilyn Spitz; 1953
Ophthalmic Laboratory
570 University Ave.
Palo Alto, Calif.
Sponsor: Dr. O. R. Tanner

May, Miss Elizabeth; 1955
Racine Orthoptic Clinic
617 Main St.
Racine, Wis.
Sponsor: Dr. William H. Bennett

Mayfield, Miss Phoebe; 1953
300 McKnight Ave.
Clayton 24, Mo.
Sponsor: Dr. S. Albert Hanser

McCullough, Mrs. Blanche M.; 1949
521 David Whitney Bldg.
Detroit 26, Mich.
Sponsor: Dr. Lester E. McCullough

- McEwen, Mrs. Mary Sisson; 1952
Address not known
- McLuckie, Mrs. Evelyn; 1955
Department of Ophthalmology
Mercy Hospital
Stevenson and Locust Avenues
Pittsburgh, Pa.
Sponsor: Dr. Robert F. Rohm
- McVay, Mrs. Marion Weygand; 1942
604 Humiston Dr.
Bay Village, Ohio
- Middleton, Miss Judith D.; 1953
717 Medical Dental Bldg.
925 Georgia St.
Vancouver 1, B. C., Canada
Sponsor: Dr. John A. McLean
- Miesel, Mrs. M. Dorothy; 1951
1613 David Whitney Bldg.
Detroit 26, Mich.
Sponsor: Dr. Duncan A. Campbell
- Miller, Mrs. Donna Speicher; 1955
4421 Main
Kansas City 11, Mo.
Sponsor: Dr. John McLeod
- Miller, Mr. Robert S., 1950
Suite 401
2040 W. Wisconsin Ave.
Milwaukee 2, Wis.
Sponsor: Dr. Samuel S. Blankstein
- Mimms, Miss Julie; 1950
Mississippi Optical Dispensary
Medical Arts Bldg.
Jackson, Miss.
Sponsor: Dr. W. Lauch Hughes
- Mittelstaedt, Miss Gloria; 1949
3000 Dickerson
Detroit 15, Mich.
- Moore, Miss Sally; 1955
Room 524
Eye Institute
635 W. 165th St.
New York 32, N. Y.
Sponsor: Dr. Maynard C. Wheeler
- Moulton, Mrs. Bonnie; 1952
5 Grove St.
Bangor, Maine
- Mulcahy, Mrs. Sharon Conlon; 1953
1085 N. Palm Ave.
Whittier, Calif.
- Murphy, Miss Jeanette; 1947
Orthoptic Clinic
Colorado University Medical Center
4200 E. Ninth Ave.
Denver 20, Colo.
Sponsor: Dr. J. L. Swigert
- Nelson, Mrs. Betty; 1945
540 Sabal Palm Dr.
Key Biscayne
Miami 49, Fla.
- Nogueira, Miss Zaida T.; 1955
Rua Sao Vicente de Paula, 416
Sao Paulo, Brazil
Sponsor: Dr. Moacyr E. Alvaro
- Nowels, Mrs. Neale; 1942
3715 Donald St.
Eugene, Ore.
Sponsor: Dr. George B. McCallum
- Ogilvie, Miss Margaret; 1956
Orthoptic Clinic
Montreal General Hospital
Montreal, Que., Canada
- O'Neill, Miss Patricia M.; 1953
1930 Chestnut St.
Philadelphia 3, Pa.
Sponsor: Dr. Edmund B. Spaeth
- Pang, Miss Stephanie; 1957
610 Steiner St.
San Francisco, Calif.
- Parry, Mrs. Vera; 1953
2804 Main St.
Buffalo 14, N. Y.
Sponsor: Dr. Joseph A. Schutz
- Patterson, Mrs. Nancy Moscrop; 1952
3215 Mathers
West Vancouver, B. C., Canada
- Payne, Miss Lydia; 1956
1816 R St.
Washington, D. C.
Sponsor: Dr. Dorothy B. Holmes
- Pellett, Mrs. Dolores Armstrong; 1953
Box 288
Brawley, Calif.
Sponsor: Dr. George Jaquith
- Peter, Mrs. Eleanor Reaves; 1948
4021 97th Ave., S. E.
Mercer Island, Wash.

Peterson, Miss Martha E.; 1943
3499 Leighton Rd.
Columbus 21, Ohio
Sponsor: Dr. Harry M. Sage

Petievich, Mrs. Zaida; 1954
Oakland Orthoptic Laboratory
447 29th St.
Oakland 9, Calif.
Sponsor: Dr. Millard E. Gump

Pick, Miss Nina; 1954
Orthoptic Clinic
Indiana University Medical Center
1300 W. Michigan
Indianapolis, Ind.
Sponsor: Dr. V. A. Teixler

Pietrini, Miss Rachael M.; 1951
8 West St.
Danbury, Conn.
Sponsor: Dr. Harold C. Patterson

Pop, Miss Alice; 1954
Orthoptic Clinic
Health Centre for Children
Vancouver General Hospital
Vancouver, B. C., Canada
Sponsor: Dr. John A. McLean

Pumphrey, Mrs. Dolores Jalbert; 1939
100 N. Main St.
Mt. Vernon, Ohio
Sponsor: Dr. Gordon H. Pumphrey

Ragan, Mrs. Ruth J.; 1947
1015 Heyburn Bldg.
Louisville, Ky.
Sponsor: Dr. C. Dwight Townes

Reimer, Miss Dorothy F.; 1951
Orthoptic Clinic
Toledo Hospital
Toledo 6, Ohio
Sponsor: Dr. R. D. Kiess

Ritter, Miss Velma; 1954
1330 Wishon Ave.
Fresno 4, Calif.
Sponsor: Dr. Richard H. Whitten

Rivera, Miss Hilda; 1955
Bureau of Crippled Children
Department of Health
Santurce, Puerto Rico
Sponsor: Dr. Guillermo Pico

Roberson, Mrs. Ruby Hall; 1948
Route 1, Box 308
Wise, Va.

Roberts, Mrs. Nancy Spitler; 1949
Address not known

Robson, Miss J. Diane; 1955
306 Vancouver Block
736 Granville St.
Vancouver, B. C., Canada
Sponsor: Dr. A. R. Anthony

Romanio, Miss Jane; 1953
Orthoptic Clinic
New York Eye and Ear Infirmary
218 Second Ave.
New York 3, N. Y.
Sponsor: Dr. Hobart A. Lerner

Rose, Miss Esme W.; 1953
Department of Ophthalmology
Ohio State University Hospital
Neil and 11th Ave.
Columbus 10, Ohio
Sponsor: Dr. William H. Havener

Rosen, Mrs. Pearl P.; 1939
692 High St.
Newark 2, N. J.
Sponsor: Dr. Emanuel Rosen

Rosenfeld, Mr. Jack M.; 1951
4032 Wilshire Blvd.
Los Angeles 5, Calif.
Sponsor: Dr. Cecelia Ross

Ross, Mrs. Florence Bateson; 1939
55 E. Washington St.
Chicago, Ill.
Sponsor: Dr. Perry W. Ross

Roth, Miss Edith V.; 1941
Evanston Hospital Assn.
Orthoptic Department
2650 Ridge Ave.
Evanston, Ill.
Sponsor: Dr. Miriam D. Eubank

Ruelle, Miss Mary; 1957
1242 Forsythe
Columbus, Ohio
Sponsor: Dr. George T. Stine

Ruzesky, Mrs. Shirley Amundsen; 1951
Division of Orthoptics
Department of Ophthalmology
University of Alberta Hospital
Edmonton, Alta., Canada
Sponsor: Dr. M. R. Marshall

- Samson, Mrs. Joan Bennett; 1952
2407 Valmont St.
New Orleans, La.
- Scharlach, Mrs. Donna Orlando; 1951
Buffalo Orthoptic Center
52 Maple St.
Buffalo 4, N. Y.
Sponsor: Dr. Howard H. Higgs
- Schuster, Miss Martha M.; 1948
Kansas City Orthoptic Clinic
248 Plaza Theater Bldg.
4711 Central
Kansas City, Mo.
Sponsor: Dr. John McLeod
- Schutte, Miss Carol; 1956
Washington University School of Medicine
640 S. Kingshighway Blvd.
St. Louis 10, Mo.
Sponsor: Dr. Bernard Becker
- Schwarz, Mrs. Ruth McNab; 1943
13210 Shaker Sq.
Cleveland 20, Ohio
Sponsor: Dr. Francis S. Schwarz, Jr.
- Sharpe, Miss Mary Glen; 1949
Orthoptic Clinic
Hahnemann Hospital
Philadelphia 2, Pa.
Sponsor: Dr. Harry S. Weaver, Jr.
- Shears, Miss Susan; 1954
Address not known
- Silva-White, Miss Lilian
c/o British Columbia House
Regent St.
London W1, England
- Simmons, Miss Barbara; 1954
311 Doctors Bldg.
478 Peachtree St., N. E.
Atlanta, Ga.
Sponsor: Dr. Alton V. Hallum
- Smith, Mrs. Charlotte Danforth; 1953
59 Argyle Park
Buffalo, N. Y.
- Smith, Miss Dorothy Mills; 1948
14 W. Mt. Vernon Pl.
Baltimore 1, Md.
Sponsor: Dr. Charles E. Iliff
- Smith, Mr. Glendon G.; 1952
Kuhn Clinic
112 Rimbach St.
Hammond, Ind.
Sponsor: Dr. Hedwig S. Kuhn
- Snell, Miss Marjorie; 1950
Orthoptic Clinic
Children's Hospital
Winnipeg, Man., Canada
Sponsor: Dr. I. H. Beckman
- Sorsby, Mrs. Mary Virginia Stallworth; 1951
Orthoptic Clinic
University of Alabama Medical Center
Birmingham, Ala.
Sponsor: Dr. Thomas O. Paul
- Speckman, Mrs. Lois Brown; 1940
109 Bayberry Lane
Levittown, L. I., N. Y.
- Spero, Mrs. Rosa deCarlo
36-35 193rd St.
Flushing, N. Y.
Sponsor: Dr. Mary G. Bruno
- Stanley, Miss Elisabeth B.; 1955
Orthoptic Department
Moorfields Eye Hospital
High Holborn
London W. C. 1, England
- Stark, Miss Elizabeth K.; 1939
30 E. 40th St.
New York 16, N. Y.
Sponsor: Dr. Harry Eggers
- Stearns, Miss Arlene E.; 1948
1652 David Whitney Bldg.
Detroit 26, Mich.
Sponsor: Dr. A. D. Ruedemann
- Stelzer, Miss Anita J., 1940
St. Louis Ophthalmic Laboratory
Beaumont Medical Bldg.
3720 Washington Blvd.
St. Louis 8, Mo.
Sponsor: Dr. Adolph C. Lange
- Stobie, Miss Dorothy; 1948
2804 Main St.
Buffalo 14, N. Y.
Sponsor: Dr. Joseph A. Schutz
- Stone, Miss Barbara; 1952
Via Guicciardini-15
Firenze, Italy
- Stromberg, Miss Ann E.; 1945
Orthoptic Clinic
Massachusetts Eye and Ear Infirmary
243 Charles St.
Boston 14, Mass.
Sponsor: Dr. E. B. Dunphy

Swenson, Miss Angela A.; 1940
Room 206
Ophthalmic Motility Clinic
905 University Ave.
Madison, Wis.
Sponsor: Dr. C. W. Aageson

Swift, Mrs. Marcella Kubilus; 1955
Department of Ophthalmology
Iowa University Hospitals
Iowa City, Iowa
Sponsor: Dr. Hermann M. Burian

Taylor, Miss B. Evelyn; 1951
708 Park Ave.
New York, N. Y.
Sponsor: Dr. Conrad Berens

Teppert, Miss Virginia; 1956
208 David Whitney Bldg.
Detroit 26, Mich.
Sponsor: Dr. Ralph Pino

Thoen, Mrs. Marguerite Madden; 1954
1024 Seventh Ave.
Lewiston, Idaho

Thompson, Mrs. Louise Altick; 1949
610 Duke St.
Norfolk, Va.
Sponsor: Dr. C. C. Cooley

Tibbs, Miss Aleatha J.; 1950
311 Doctors Bldg.
478 Peachtree St., N. E.
Atlanta, Ga.
Sponsor: Dr. F. Phinzy Calhoun

Tomlinson, Miss Evelyn; 1957
16 Hermann Professional Bldg.
6419 Fannin St.
Houston 25, Texas
Sponsor: Dr. Louis J. Girard

Toney, Miss Helen F.; 1953
105 Hawes St.
Wharton, Texas
Sponsor: Dr. Vernon A. Black

Travers, Mrs. Elsie Schatzle; 1952
112 Third St.
Cohoes, N. Y.

Ulich, Mrs. Ann Wright; 1953
Boston City Hospital
Dowling 2
Boston, Mass.
Sponsor: Dr. D. R. Alpert

Updegraff, Mrs. Ramona; 1953
911 Rider
Iowa City, Iowa

Urist, Mrs. Pearl T.; 1944
432½ Phoenix St.
South Haven, Mich.
Sponsor: Dr. Martin J. Urist

Wackerhagen, Miss Mary; 1954
115 E. Capital Ave.
Little Rock, Ark.
Sponsor: Dr. Dale Alford

Wahlgren, Miss Ruth; 1953
Department of Ophthalmology
University of Oregon Medical School
Portland, Ore.
Sponsor: Dr. Kenneth C. Swan

Walraven, Miss Frances C.; 1939
235 Strickler Bldg.
1293 Peachtree St., N. E.
Atlanta 9, Ga.
Sponsor: Dr. J. Mason Baird

Ward, Miss Jane R.; 1945
Department of Ophthalmology
Cleveland Clinic
2020 E. 93rd St.
Cleveland 6, Ohio
Sponsor: Dr. Roscoe J. Kennedy

Wehrheim, Miss Dixie; 1952
Room 512
Medical Arts Bldg.
1211 21st Ave., S.
Nashville 12, Tenn.
Sponsor: Dr. George W. Bounds, Jr.

Weingeist, Mrs. Fausta; 1956
180-33 80th Dr.
Jamaica 32, L. I., N. Y.
Sponsor: Dr. Samson Weingeist

White, Mr. N. LeRoy; 1949
228 Medical Arts Bldg.
Portland 5, Ore.
Sponsor: Dr. E. Merle Taylor

Whiteford, Mrs. Beatrice Chantler; 1955
Apt. 5
804 N. Dubque
Iowa City, Iowa

Wilkinson, Mrs. Mary Caston; 1953
85 Sanford St.
Buffalo, N. Y.

Williams, Mrs. Barbara Coder; 1954
616 Church St.
Ann Arbor, Mich.

Williams, Mrs. Marie F., 1947
University of Colorado Medical Center
4200 E. Ninth Ave.
Denver 20, Colo.
Sponsor: Dr. Herbert M. Katzin

Wood, Miss Dorothy; 1956
1605 22nd St., N. W.
Washington, D. C.
Sponsor: Dr. M. Gordon Anderson

Woodward, Jr., Mr. Fletcher D.; 1954
9 S. E. 15th Ave.
Fort Lauderdale, Fla.
Sponsor: Dr. Frank D. Costenbader

Worsham, Mrs. Marguerite Johnson; 1947
161 W. Cheves St.
Florence, S. C.
Sponsor: Dr. J. Howard Stokes

Young, Mrs. Barbara Behn; 1952
1227 Hillside
Austin 4, Texas

Ziel, Mrs. Kathleen Byers; 1948
Grand Rapids Orthoptic Clinic
338 Sheldon, S. E.
Grand Rapids, Mich.
Sponsor: Dr. Ralph H. Gilbert